

INTRACRANIAL
COMPLICATIONS
OF
EAR, NOSE AND
THROAT INFECTIONS

INTRACRANIAL COMPLICATIONS OF EAR, NOSE AND THROAT INFECTIONS

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TO
THE MEMORY OF MY FATHER
BERTHOLD BRUNNER

Preface

For a quarter of a century, as clinician and teacher, I have studied the intracranial complications of ear, nose and throat infections. This work was begun under the guidance of Prof. Gustav Alexander and carried on after the death of that remarkable man.

This book is not intended to serve as a textbook of otorhinology. It is assumed that the reader is acquainted with the principles of both otorhinology and neurology.

The view that intracranial complications of ear, nose and throat infections no longer are a significant problem is unsound. Admittedly, the incidence of such complications has decreased. Nevertheless, even though a striking reduction of intracranial complications is attributable to modern chemotherapy, one must remember that a definite decrease in incidence had been noted years before the advent of chemotherapy. Chemotherapy is of definite value in the treatment of intracranial complications; its influence in the prophylaxis of these complications is controversial. However, it is a fact that not infrequently chemotherapy of the primary condition alters the customary symptomatology and renders diagnosis and treatment of intracranial complications more difficult than before the introduction of these new therapeutic agents. Thus we are dealing in this volume with problems that continue to deserve the attention of the otolaryngologist and neurologist.

Most of my earlier work was done while engaged in post-graduate teaching in Vienna before the war; therefore some of

my important records are not available. Of those I was able to preserve I have made free use in this volume. The same circumstances are responsible for the absence of a great number of statistics. Their lack, however, does not seem too important, because statistics are frequently more confusing than enlightening, particularly those based on a review of the literature. Furthermore, pertinent statistics have already been published, as reference to the *Quarterly Cumulative Index Medicus* and the splendid summaries of intracranial complications by Wells P. Eagleton will show; their repetition here would be unnecessary. It is almost inevitable that certain important studies escape attention when reviewing the literature. Such omissions are not intentional. I have deliberately avoided overloading the following pages with quotations and an elaborate bibliography and have included only references which offer an exhaustive review of the pertinent literature.

I am indebted to numerous persons for advice and aid in the preparation of this work. Without the generous efforts of Dr. F. L. Lederer and Dr. E. LeRoy Wood the writing of this work might never have been possible. Dr. A. R. Hollender gave special impetus to the preparation of the volume and constant encouragement. Drs. Percival Bailey, A. J. Coombs, G. S. Livingston, S. M. Morwitz, I. Spiesman and O. E. Van Alyea read portions of the manuscript and offered helpful criticisms. Dr. L. Bergmann made special anatomic studies for several illustrations. Special mention is also due Mr. Tom Jones, Mrs. Maria Ikenberg, Miss Angela Bartenbach and Mr. L. A. Toriello for their contributions to the illustrative material used here, and also the publisher for constant co-operation.

—HANS BRUNNER.

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SECTION I

General Introduction

Anatomy and Physiology

DURA MATER (PACHYMEINIX)

THE cerebral dura consists of (1) connective tissue, (2) blood vessels, (3) nerves, (4) lymphatic crevices and (5) mesothelium. The connective tissue is firm and poor in cells. An internal and external layer can be distinguished if the fibers of each run in a different direction. The external layer serves as the internal periosteum of the bones of the skull. In this layer are found the ramifications of the middle meningeal artery, originating from the internal maxillary artery; the anterior meningeal artery, originating from the anterior ethmoid artery, and the posterior meningeal artery, originating from the pharyngeal ascending artery. Only the branches of the middle meningeal artery run in sulci on the inside of the skull which can be visualized on x-ray pictures.

The blood vessels of the dura form an external and an internal network. The external network consists of branches of the meningeal arteries which frequently anastomose with one another and are usually accompanied by two veins. The internal network contains fewer blood vessels than the external layer and consists of venous capillaries which anastomose with one another and frequently present saclike dilatations (Fig. 29, pp. 92 f.). There are numerous anastomoses between the two networks (Fig. 32, p. 99): fine arterial capillaries originating from the arteries of the external network run through the dura and empty into the dilatations of the internal capillary bed, and veins of the internal network run toward the veins of the external

network. The blood of the pachymeninx is carried away by the meningeal veins and dural sinuses. It must be emphasized that the arteries of the dura are found principally in the external layer, whereas the venous capillaries occur chiefly in the internal layer. This arrangement probably contributes to the considerable resistance of the dura to infections which originate in the adjacent tympanic cavity and paranasal sinuses.

The external network of the dura anastomoses with the blood vessels of the bone and the mucosa of the tympanic cavity and paranasal sinuses (Fig. 5, p. 15). The internal network communicates with the blood vessels of the pia-arachnoid. The arteries of the external network have fine branches which carry blood into the bone. Other branches break up into capillaries within the mucosa of the tympanic cavity and paranasal sinuses. The venous capillaries leave the mucosa along the same channels which lead the arterial capillaries into the mucosa and drain into the veins of the external network. The anastomosing veins are valveless, and thus the blood may flow in either direction. These anastomosing arterial and venous blood vessels which run between the dura and the mucosa of the tympanic cavity and paranasal sinuses are important in the pathogenesis of intracranial complications.

The nerve supply of the dura is furnished chiefly by the three divisions of the trigeminal nerve. The dura of the posterior fossa is innervated principally by branches of the vagus nerve and probably by branches of the glossopharyngeal and the three upper cervical nerves. The dura contains many branches of the sympathetic nerve, probably originating from the sympathetic nerves in the carotid canal and running parallel to the meningeal arteries. Both the anterior third of the superior longitudinal sinus and the cavernous sinus are supplied by the first division of the trigeminal nerve. The posterior third of the superior longitudinal sinus, the transverse sinus and the superior petrosal sinus are crossed by many branches of the nervus tentorii (nervus recurrens of Arnold). This nerve leaves the first division of the trigeminal, turns back and runs in, or close to, the sheath of the

trochlear nerve. Near the attached border of the tentorium its fibers leave the trochlear sheath and spread out fanwise within the tentorium. The nerve fibers reach the walls of the aforementioned sinuses and are particularly numerous in the region of the torcular. Here they turn upward into the falx and can be followed forward for some distance. In the walls of the sigmoid sinus and jugular bulb may be seen fine nerve fibers, supplied by the ninth and tenth nerves in the jugular foramen. It is a moot question to what extent the sinuses are actually innervated by the nerves which cross them. No nerve endings have been found in the sinus walls. The dura is not sensitive to pain, except parts at the base of the skull, but sensitivity to pain is present in the arteries of the dura (except the small terminal branches), the cerebral arteries at the base of the brain, the fifth, ninth and tenth cranial nerves, the three upper cervical nerves and, to a lesser degree, the great venous sinuses and their tributaries from the surface of the brain.

Certain typical patterns of pain reference originating in the dura are of interest to the otolaryngologist. The anterior portion of the falx and longitudinal sinus refer pain to the forehead and nose. The lateral sinuses may refer pain along the recurrent nerve (Arnold) forward to the back of the ipsilateral eye. Reference of pain from the posterior cranial fossa is apt to be to the ipsilateral mastoid or occiput and neck.

The dura is tunneled by a system of crevices which are believed to communicate with the subdural spaces and to be continuous with lymph vessels which commence on the external surface of the dura and extend to the neighboring lymph nodes. However, a free anastomosis between the crevices of the dura and the lymph nodes apparently does not exist since neither in sinus thrombosis nor in extensive pachymeningitis are inflamed lymph nodes found. Several authorities state definitely that the dura does not contain true lymphatic vessels, i.e., vessels possessing an endothelial lining.

On the inside of the dura the mesothelium forms a thin layer which is quite resistant to infection. The mesothelium occasionally consists of two layers. Usually it is very delicate, particularly in

adults, so that it is visualized with difficulty, even microscopically.

Whereas in adults the dura can be stripped easily from the vault of the skull, there are firm attachments between dura and sutures and fontanels in the newborn and in children up to 10 years. At the base of the skull the findings differ at various sites (Fig. 1). The anterior and posterior surfaces of the petrous bone and the temporal squama are covered by dura. At the posterior surface of the petrous bone the dura is firmly attached in the area of the internal auditory meatus, cochlear aqueduct, subarcuate fossa and vestibular aqueduct. The dura covers the internal auditory meatus up to the fundus of the canal (Fig. 16, p. 39) and enters the cochlear aqueduct to the periosteum of the scala tympani. In the newborn the subarcuate fossa forms a large cavity in the petrous bone. The roof of the cavity is formed by the eminentia arcuata and the cavity itself is filled with blood vessels and connective tissue deriving from the dura. Later in life, bone grows at the opening of the groove and ultimately causes an osseous obliteration of the groove. However, even in adults, microscopic examination discloses blood vessels and connective tissue, deriving from the dura, at the site of the subarcuate fossa. Occasionally there is a retroarcuate fossa consisting of a slit in the bone lateral to the vestibular aqueduct. The slit is filled with connective tissue originating from the dura. Within the vestibular aqueduct runs the endolymphatic duct which forms the endolymphatic sac, embedded within the dura (Fig. 39, p. 130). Likewise, there is a firm attachment of the dura to the superior petrous sulcus for 1 cm. or more lateral to the internal auditory meatus. At several sites on the posterior surface of the petrous bone the dura is very thin, particularly in the angle between the lateral sinus and the petrosal superior sinus. On the anterior surface of the petrous bone the dura forms numerous strands of connective tissue and blood vessels which extend into the tegmen tympani and the foramen lacerum. These strands are more numerous in infants than in adults, they are also more numerous when there is hyperplastic mucosa in the tympanic cavity than when the tympanic mucosa is normal.

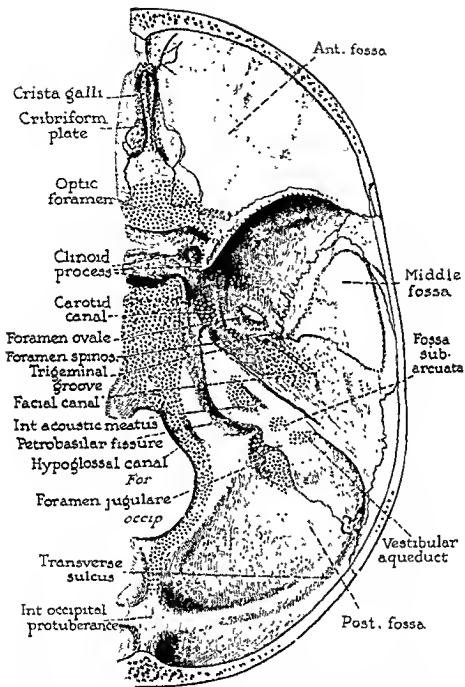


FIG. 1.—Base of the skull. Dotted areas indicate sites of firm attachment of the dura to the base of the skull. (Modified from A. E. Walker, *Anat. Rec.* 55:291, 1933.)

The anatomy of the dura at the apex of the petrous bone is of great importance. In textbooks the findings are frequently described as follows. The dura of the middle cranial fossa forms a duplication at the apex of the petrous bone, called the cavity of Meckel. Within this cavity is embedded the crescent-like gas-

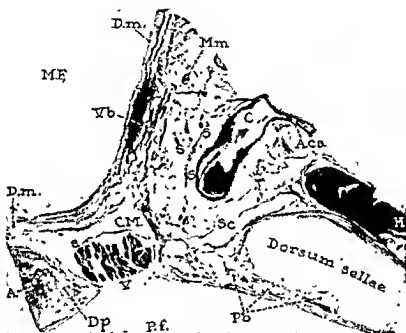


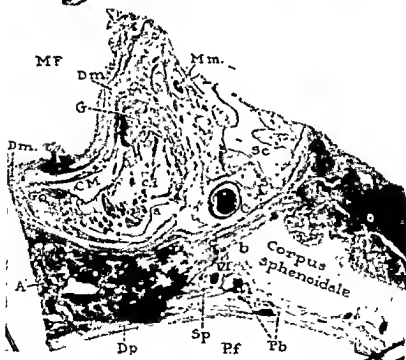
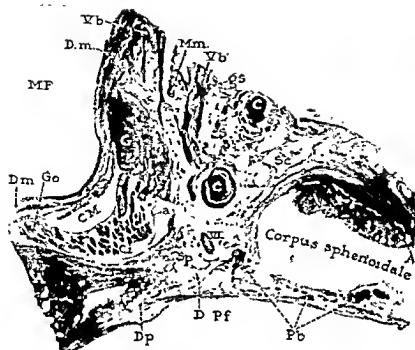
FIG. 2A—Oblique horizontal section through apex of the petrous bone (A), dorsum sellae (cartilaginous), hypophysis (H), cavernous sinus (Sc) and internal carotid artery (C) in the newborn Pf, posterior cranial fossa, Mf, middle cranial fossa, Dp, dura of posterior fossa, Dm, dura of middle fossa, V, root of trigeminal nerve covered by an arachnoid sheath (a), CM, cavum mecklii, Pb, basilar plexus anastomosing with cavernous sinus, Aca, anterior cerebral artery, Vb, second branch of trigeminal nerve, x, branches of sympathetic nerve; Mm, branch of middle meningeal artery.

serian ganglion which receives the three divisions of the trigeminal nerve at its convexity and discharges the roots of this nerve at its concavity. The entire structure occupies the impressio trigemini. This description is not correct. The impressio trigemini harbors the roots of the trigeminal nerve, but only the occipital pole of the gasserian ganglion. If the petrous apex is short and does not extend to the gasserian ganglion, the major portion of

the ganglion lies on tendon-like connective tissue. This, being a part of the dura, extends from the petrous apex to the mesial margin of the foramen ovale and separates the internal carotid from the gasserian ganglion (Fig. 2, B and C). If the petrous apex is long, it advances between the gasserian ganglion and the carotid artery, separating these structures. In either case the dura extends into the carotid canal and forms the periosteum of the canal.

Furthermore, Meekel's cavity is not formed by a duplication of the dura of the middle cranial fossa. Rather, it is an evagination of the dura of the posterior cranial fossa under the dura of the middle cranial fossa. Therefore, if the dura of the middle cranial fossa is cautiously stripped from Meekel's cavity, a sac-like diverticulum, originating in the dura of the posterior fossa, makes its appearance. The opening of the diverticulum presents the dural slit, allowing the entrance of the trigeminal roots. The superior wall of the sac covers the roots of the trigeminal nerve and the ganglion; the inferior wall is the periosteum of the impressio trigemini and extends into the carotid canal. At the distal boundary of the gasserian ganglion the wall of the sac passes into the epineurium of the trigeminal divisions. The dural sac separates the roots of the trigeminal nerve and the ganglion from both the cavernous sinus and the canal of Dorello. There is no connection between the cavernous sinus, which is embedded in the dura of the middle fossa, and Meekel's cavity, which contains a diverticulum of the dura of the posterior fossa (Fig. 2A).

Below and mesial to the trigeminus, the abducens nerve pierces the dura and enters the canal of Dorello (Fig. 2B), which has the following structure. The sulcus petrosus superior comes to an end 4-5 mm. proximal to the apex of the petrous bone and is there replaced by a bony process, the tip of which is directed toward the posterior clinoid process. This process, called the sphenoid spine, is the continuation of the posterior lamella of the superior petrosal sulcus. The superior petrosal sinus bends anteriorly, outward from the sphenoid spine, to run into the cavernous sinus; it has no contact with the petrous apex. Below



the posterior clinoid process is another small bony process which turns outward, called the accessory process of the posterior clinoid (Fig. 2C). Between this process and the sphenoid spine runs the petrosphenoid ligament (Fig. 2C), which consists of the deepest bundles of the tentorium. Thus a small, approximately three-sided, space is formed, with the apex outward and with the base directed inward. Through this space, the canal of Dorello, run the abducens nerve and the inferior petrosal sinus (Figs. 2B and 2C). The nerve is covered by an arachnoid sheath and is sometimes closer to the lumen of the inferior petrosal sinus, sometimes closer to the petrous bone. Occasionally a pacchionian tuft enters the canal with the nerve and bulges into the inferior petrosal sinus. On leaving Dorello's canal the abducens nerve makes a slight bend and enters the cavernous sinus.

In the area of the temporal squama there are no firm attachments between dura and bone in adults; in the newborn and in infants the attachment is more firm.

In the region of the nasal cavities the dura is loosely attached to the roof of the ethmoid. In the newborn, broad connective tissue strands extend into the sphenoid body and connect the mucosa of the nasopharynx with the dura of the planum sphenoidale. When the development of the sphenoid sinus is completed, the connective tissue strands become ossified, but the firm attachment between dura and planum sphenoidale persists even in adults. Likewise, the dura is firmly attached to the crista galli, around the foramina of the cribriform plate (Fig. 17, p. 41) and the ethmoid foramina, which open into the orbit. There is also a firm attachment between dura and clinoid process.

In front of the crista galli is the foramen cecum (Fig. 5) which in adults forms a culdesac and contains a few connective

← FIG. 2B (above).—Section lower than that in Figure 2A. *C*, gasserian ganglion; *Go*, occipital pole of gasserian ganglion; *D*, Dorello's canal; *VI*, abducens nerve; *Sp*, sinus petrosus inferior; *gs*, sympathetic ganglion; *VI'*, first branch of trigeminal nerve; *A*, ossification of sphenoid body; *Cl*, cisterna of gasserian ganglion. Other abbreviations as in Figure 2A.

FIG. 2C (below).—Section lower than that in Figure 2B. *b*, accessory process of posterior clinoid; *d*, petrosphenoid ligament; *VI*, abducens nerve. Other abbreviations as in Figure 2A.

FIG. 4—Principal veins of brain and face and their anastomoses with dural sinuses. 1, supra-orbital vein, 2, frontal diploic vein, 3, anterior ethmoid vein, 4, vein from olfactory bulb, 5, veins from dura, 6, posterior ethmoid vein, 7, infra-orbital vein, 8, sphenopalatine vein from nasal mucosa to pterygoid plexus; 9, superficial temporal vein, 10, rete foraminis ovalis, 11, foramen spinosum, 12, middle meningeal vein, 13, sphenoparietal sinus; 14, veins from tympanic cavity, antrum and mastoid, 15, veins from tympanic cavity; 16, petrosquamous sinus, 17, vein of Labbé, 18, ophthalmopetrous sinus, 19, middle and inferior cerebral veins, 20, veins from tentorium, 21, superior petrosal sinus, 22, vena forae subarcuatae, 23, vena aquaeductus vestibuli; 24, veins from tympanic cavity, 25, vena auditiva interna, 26, vena aquaeductus cochleae; 27, plexus basilaris, F. ethmoid (After Macewen, Turner and Reynolds.)

tissue strands deriving from the dura. In the newborn and infants these connective tissue strands pass through the foramen caecum and serve as an internal periosteum of the bones at the back of the nose.

SINUSES OF THE DURA MATER

The sinuses are channels within the dura which anastomose with each other. The walls consist of firm connective tissue con-

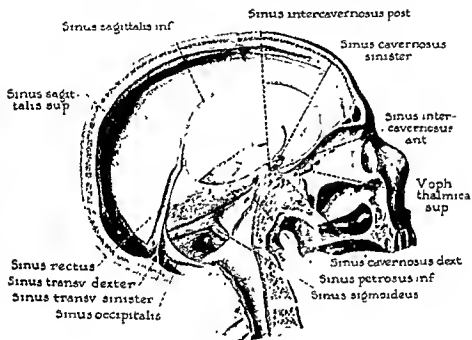


FIG. 3—Dural sinuses (After Tandler)

taining several capillaries and arterioles. There is some elastic tissue but no muscular tissue. The endothelium consists of a single layer of flat cells. The sinuses do not contain valves. Figure 3 presents the sinuses in the skull in typical cases, and Figure 4, the chief anastomoses of the sinuses. This discussion deals only with the sinuses which are important to the otolaryngologist.

Superior longitudinal sinus.—This sinus originates in the area of the foramen caecum where the falx cerebri is split into two layers which lateralward gradually pass into the dura of the

anterior fossa. Thus the falx forms a tentlike space from which rises the superior longitudinal sinus.

In the newborn the sinus anastomoses with veins of the periosteum and perichondrium of the dorsum nasi in the midline and with the nasal veins lateral to the midline (Fig. 5A). Later in life the frontal sinns separates the two venous systems (Fig. 5B), and the anastomosis between the superior longitudinal sinus and the veins of the dorsum nasi becomes obliterated. As remnants of the original anastomosis, there are, in adults, small veins which originate in the mucosa of the frontal septum, perforate the posterior wall of the frontal sinus and drain into the superior longitudinal sinus. In infants, and occasionally in adults, there is a second anastomosis between the sinus and the nasal veins. The small veins of the nasal septum and lateral wall of the nasal cavity pass through the anterior foramina of the cribriform plate with olfactory fibers to form a venous plexus within the dura and between dura and bone (Fig. 5A, *Pl.*) which anastomoses with the superior longitudinal sinus. One of these veins, the vein of Zuckerkandl, originates in the mucosa of the agger nasi, passes through the cribriform plate and anastomoses with the venous plexus above the cribriform plate or with a vein at the orbital surface of the frontal lobe which, in turn, drains into the superior longitudinal sinus. Another vein, the anterior ethmoid (Fig. 4), originates in the ethmoid area, anastomoses with the superior ophthalmic vein and drains into the venous plexus above the cribriform plate. There are also anastomoses between the superior longitudinal sinus and small veins of the dura, falx and olfactory bulb and tract.

The superior longitudinal sinus runs backward, immediately below the vault of the skull, in the superior margin of the falx. It has a triangular lumen, increasing in size toward the torcular. In infants the increase in size is rather abrupt just behind the posterior angle of the anterior fontanel. Within the lumen are several strands of connective tissue which conceal some of the openings of the superior cerebral veins into the sinus. In rare instances the anterior portion of the sinus presents

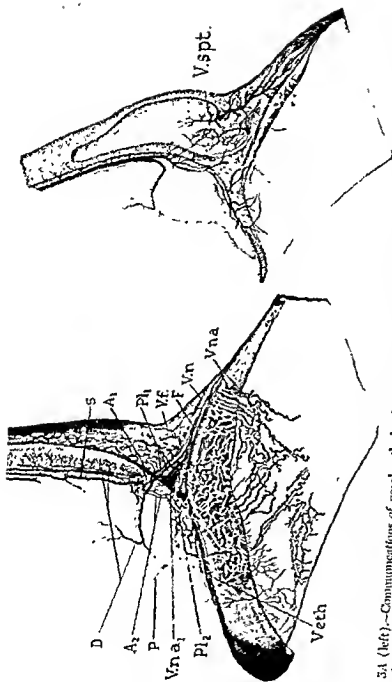


Fig. 5A (left).—Communications of nasal with dural veins in newborn. *S*, longitudinal superior sinus; *A*₁, asymmetrical anastomosis; *D*, veins of dura; *Pl*₁, venous plexus in foramen caecum; *F*, veins at anterior boundary of anterior lateral nasal veins; *Veth*, external pterosteal veins of frontal bone; *Vna*, anterior mesial and interfrontal septum; *Vna*₁, veins of nasal mucosa.

Fig. 5B (right).—Anastomoses between veins of frontal sinus with dural veins and sinuses in adult. *V*_{opt}, veins of mucosa of interfrontal septum. (After Drechsel, *Ztschr. f. Anat. u. Entwicklungsgesch.* 90:57, 1929.)

saclike protrusions which perforate the frontal bone and form compressible tumors in the area of the bridge of the nose, called sinus pericranii.

At the protuberantia occipitalis the sinus anastomoses with the sinus rectus and the lateral sinus. There is great variability in the anastomoses at this site. Usually the superior longitudinal sinus drains into the right lateral sinus and anastomoses with the left lateral sinus only by a small blood vessel. Some anatomists state that in 50 per cent of specimens the superior longitudinal sinus is directly continuous with one of the lateral sinuses at the torcular. In that case it is continuous with the right lateral sinus in three of four cases. Several theories (reviewed by Woodhall) have been advanced to explain the tendency of the superior longitudinal sinus to anastomose with the right rather than with the left lateral sinus.

Particularly over the central region of the convexity of the brain there are irregularly formed cavities, called lateral venous lacunae (Fig. 11), within the dura on both sides of the sinus. These lacunae anastomose with the sinus by narrow channels and occasionally extend up to 3 cm. laterally from both sides of the sinus. They are usually small over the frontal and occipital lobes and large over the parietal lobe. They receive blood from the dura and diploe, whereas the superior cerebral veins usually drain into the sinus and not into the lacunae. The superior cerebral veins are frequently united into four main trunks, frontal, precentral, postcentral and occipital veins. The frontal veins enter the sinus at nearly right angles and the precentral and postcentral veins run obliquely forward (Fig. 4). These veins drain the superior and mesial aspects of the cerebral hemisphere. The precentral and postcentral trunks usually anastomose with the middle cerebral and the sylvian vein, respectively, which are tributaries of the cavernous, superior petrosal or lateral sinus. Hence the superior longitudinal sinus is the chief channel draining the superior and mesial surfaces of the cerebral hemisphere. In addition, the sinus anastomoses with the external veins of the skull through one or two parietal emissaria.

Lateral sinus.—This sinus, consisting of the *transverse* and *sigmoid sinuses*, runs between two connective tissue layers which fuse medially, forming the *tentorium cerebelli*. At the superior angle of the petrous bone the sinus forms the “superior knee” and passes into the sigmoid sinus, which occupies the sigmoid sulcus of the temporal bone (Fig. 3). Near the jugular foramen

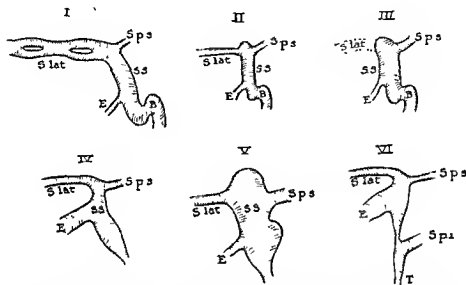


FIG. 6.—Variations of lateral sinus. *S lat*, lateral sinus; *Sps*, superior petrosal sinus; *Ss*, sigmoid sinus; *E*, emissarium mastoideum; *B*, jugular bulb; *Spi*, inferior petrosal sinus; *T*, jugular vein. In II and III, blood flows from the superior longitudinal sinus either into the lateral sinus of the other side or into a dilated occipital sinus. In V, the lateral sinus forms a diverticulum which may extend to the middle cranial fossa. In IV and VI, note compensatory dilatation of the emissarium mastoideum. Formations like III and VI are usually found on the left side.

the sinus narrows, forms the “inferior knee” and passes into the jugular bulb. Occasionally venous lacunae are found lateral to the transverse sinus. There are many variations of the lateral sinus, particularly on the left side, where it may be very narrow. Comparatively frequent variations are shown in Figure 6.

The following veins drain into the lateral sinus.

1. Several veins from the diploe.
2. Veins originating in the tympanic cavity, the mastoid antrum and the mastoid cells and draining particularly into the superior knee.

3. The mastoid vein. This vein originates in the posterior wall of the sigmoid sinus, passes through the emissarium mastoideum and drains into the posterior auricular, occipital or external jugular vein or, occasionally, the internal jugular vein. The emissarium mastoideum is usually located at the posterior boundary of the mastoid process about 1.5 cm. below the superior knee of the sinus and has, as a rule, a diameter of 0.2–0.5 cm. Frequently the emissarium can be seen

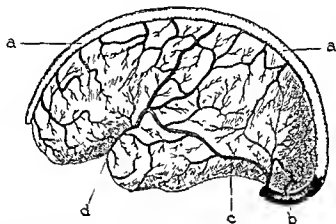


FIG. 7.—Anastomotic vein of Labbé (c) and great anastomotic vein of Trolard (d) a, superior longitudinal sinus, b, lateral sinus.

on the x-ray film. In some instances the emissarium is not present; in others there are two to four emissaria. In children the emissarium has a relatively large diameter and may reach the size of the internal jugular vein.

4. The superior and lateral veins of the cerebellum, superior and mesial veins of the cerebellum and veins originating on the inferior surface of the cerebellum, pons and medulla oblongata.
5. Veins originating in the adjacent dura and tentorium cerebelli.
6. Veins originating in the occipital lobe and other parts of the cerebrum. Of particular importance is the anastomosis of the lateral sinus with the veins of the sylvian fissure, which is

accomplished by the vein of Labbé. The anastomotic vein of Labbé (Fig. 7; see also Fig. 19, p. 56) originates in the upper knee of the lateral sinus and passes over the surface of the temporal lobe to the middle cerebral veins, which in turn anastomose with Trolard's vein (Fig. 7). The great anastomotic vein of Trolard originates on the lateral surface of the cerebral hemisphere or in the superior longitudinal sinus, passes through the sylvian fissure, pierces the dura at the margin of the lesser sphenoid wing and drains into the petrosal or the sphenoparietal sinus.

7. The internal auditory vein (in rare instances).

8. Infrequently there are venous anastomoses between the anterior wall of the bony part of the external auditory canal and the transverse sinus, between the outer surface of the mastoid and the superior knee and between the ophthalmic vein and the transverse sinus by means of an ophthalmopetrosal sinus.

The wall of the sinus adjacent to the mastoid cells is comparatively thin in the area of the sinus knees, particularly at the inferior knee, and is thicker between the knees. The wall adjacent to the cerebellum has uniform thickness and contains a few arterioles and veins. The opposite wall contains a large number of arterioles and dilated veins, which may give the impression of cavernous spaces and which drain into the sinus.

In the newborn the sigmoid sulcus is either very shallow or absent. The sigmoid sinus is covered by a bony plate about 5 mm. thick. The posterior boundary of the sinus frequently extends to the occipitomastoid suture. In general, the distance between the sigmoid sinus and the posterior wall of the external auditory canal in the newborn is about 1.5 cm.; the same distance in adults, measured at the level of the base of the mastoid process, is frequently 3 cm.

When visualized from the posterior cranial fossa, the sigmoid sulcus in adults may be shallow or deep. If shallow, it bulges only a little toward the mastoid cells and is distant from both the autrum and the cortex of the mastoid. If deep, the sinus may

extend close to both the cortex and the posterior wall of the external auditory canal—**anterior position of the sinus**. In these instances usually the dura of the middle cranial fossa also bulges toward the mastoid antrum. This renders surgical exposure of the antrum difficult. The varying depths of the sigmoid sulcus are particularly dependent on (1) the width of the sigmoid sinus, (2) the pneumatization of the mastoid process and (3) the form of the skull. Since the superior longitudinal sinus commonly drains into the right lateral sinus, the right sigmoid sinus is usually larger than the left, and for this reason the sigmoid sulcus is usually deeper on the right side. Furthermore, the anterior position of the sigmoid sinus is more common with diminished pneumatization of the mastoid and in brachycephalic skulls than with normal pneumatization and in dolichocephalic skulls. The anterior position of the sinus can be visualized on the x-ray film. Occasionally it is even possible to visualize abnormalities of the lateral sinus on the film.

Jugular bulb.—This occupies the venous portion of the jugular foramen, which is separated from the pars nervosa by the petro-occipital ligament. The extension of the bulb is dependent on the width and curvature of the sigmoid sinus. If the sinus is in a posterior position and the curvature is not marked, the sinus passes into the jugular bulb without forming a definite bulb. If the sinus is in an anterior position and the curvature is marked, the jugular bulb is large. In such instances the dome of the bulb may extend up to the level of the round or even the oval window in the tympanic cavity. It may show dehiscences toward the tympanic cavity, posterior cranial fossa, posterior semicircular canal or facial canal. Occasionally the mucosa of the tympanic cavity bulges through such a dehiscence and forms a small diverticulum between the bone of the jugular dome and the jugular bulb.

The following veins pour into the jugular bulb or into the jugular vein.

1. The inferior petrosal sinus.
2. The veins of the cochlear aqueduct.

3. The anterior condyloid veins, which run in the canal of the hypoglossal nerve and anastomose with the spinal plexus on

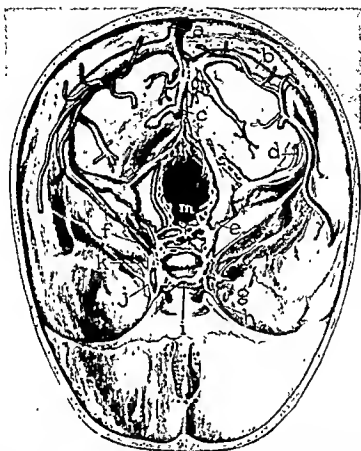


FIG. 8.—*a*, longitudinal superior sinus, *b*, transverse sinus, *c*, straight sinus, *d*, emissarium mastoideum, *e*, inferior petrosal sinus, *f*, superior petrosal sinus; *g*, emissarium foraminis ovalis; *h*, ophthalmic vein, *i*, intercavernous sinus, *j*, cavernous sinus, *k*, carotid sinus, *m*, basilar plexus (After Zuckerkandl.)

the one hand and with the jugular bulb or the internal jugular vein on the other.

4. The posterior condyloid vein, which usually runs in the condyloid canal and anastomoses with the vertebral plexus and deep veins of the neck as well as with the jugular bulb or the internal jugular vein.

Cavernous sinus.—This sinus (Figs. 2, 3, 8-10) differs from the others in two respects: the lumen is crossed by strands of

connective tissue which are thicker in children than in adults, and the sinus contains the internal carotid artery and several cranial nerves. So far as the nerves are concerned (Figs. 2 and 9), the oculomotor and trochlear nerves run close to the dura in the upper and lateral corner of the sinus; the first, or ophthal-

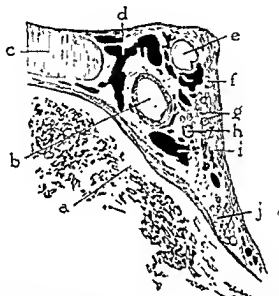


FIG. 9.—Cross-section through cavernous sinus. *a*, sphenoid body; *b*, internal carotid artery, *c*, hypophysis, *d*, cavernous sinus; *e*, oculomotor nerve; *f*, trochlear nerve; *g*, ophthalmic nerve; *h*, abducent nerve, *i*, cavernous sinus, *j*, maxillary nerve. (After Corning.)

mic, division of the trigeminus slightly below the trochlear nerve, and the second, or maxillary, division of the trigeminus below and lateral to this. The nervus abducens is close to the lateral wall of the carotid and separated from it by connective tissue. Between the bundles of the second division of the trigeminus is much connective tissue, causing marked scattering of the nerve bundles. Sympathetic ganglions are close to the ophthalmic nerve. Amyelinic nerve fibers pass with arterioles from the ganglions to the sympathetic plexus which surrounds the carotid artery.

The cavernous sinuses anastomose with each other by veins,

called *sinus intercavernosus*, which run archlike anteriorly and posteriorly to the sella tureica within the diaphragm of the sella (Fig. 8). Furthermore, small veins at the inferior surface of the hypophysis anastomose with both cavernous sinuses.

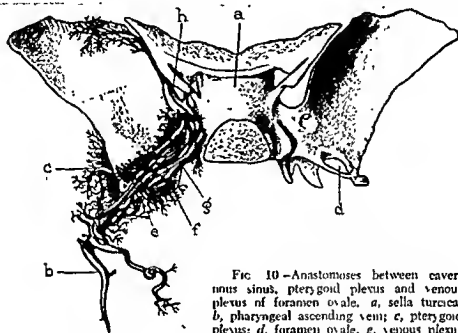


FIG. 10—Anastomoses between cavernous sinus, pterygoid plexus and venous plexus of foramen ovale. a, sella turcica, b, pharyngeal ascending vein; c, pterygoid plexus; d, foramen ovale, e, venous plexus of foramen ovale, f, cavernous sinus, g, cavity for internal carotid artery; h, ophthalmic vein (After Hochstetter.)

The cavernous sinus anastomoses with the following veins (Fig. 4).

1. The sinus sphenoparietalis, which communicates with the middle meningeal veins and is situated in the dura on the under surface of the lesser wing of the sphenoid bone.
2. The superior ophthalmic vein, which drains into the anterior portion of the sinus. Since the inferior ophthalmic vein, which anastomoses with the pterygoid plexus and the anterior facial vein, pours into the superior ophthalmic vein, there is considerable anastomosis between the cavernous sinus and the veins of the face and neck. The ophthalmic veins lack valves, and for this reason a blood flow in either direction is possible in these veins.

3. The central vein of the retina, which frequently drains into the superior ophthalmic vein.
4. Veins originating in the mucosa of the sphenoid and passing through the sphenoid bone.
5. The basilar plexus, which lies on the clivus and pours into the posterior portion of the sinus.
6. Veins which pierce the foramen lacerum, anastomose with the veins of the pterygoid plexus and drain into the posterior portion of the cavernous sinus.
7. Veins which run through the foramen ovale and create a communication between the pterygoid venous plexus and the cavernous sinus.
8. Middle meningeal veins.
9. Veins which pass through the vesalian foramen and join the pterygoid venous plexus.
10. Middle cerebral veins, which drain the blood from the inferior surface of the frontal and temporal lobe and from the posterior horizontal limb and the stem of the lateral fissure of Sylvius.
11. Veins of the tentorium cerebelli.
12. Sinus caroticus (carotid sinus, Fig. 8 and Fig. 55, p. 222), which shows essentially the same structure as the cavernous sinus and must be considered a continuation of the cavernous sinus posteriorly and inferiorly. The carotid sinus encircles the ascending and horizontal portion of the internal carotid in the temporal bone. The venous spaces which freely anastomose with the veins of the marrow of the apex of the petrous bone are embedded in firm connective tissue. Also in the carotid sinus is the nervus caroticus, which originates from the ganglion cervicale supremum. Entering the carotid sinus, the nerve divides into an anterior and a posterior branch. At the knee of the carotid artery both branches form a nervous plexus which accompanies the artery to the cavernous sinus. There are several sympathetic ganglions within the carotid sinus. Toward the internal jugular vein the sinus forms a venous plexus which ultimately drains into the internal jugular vein.

Between the lateral sinus and jugular bulb and the cavernous sinus are two sinuses: the sinus petrosus superior, and the sinus petrosus inferior. The *superior petrosal sinus* (Fig. 8) carries the blood from the cavernous sinus to the lateral sinus. It is embedded in the insertion of the tentorium at the superior angle of the petrous bone and runs back from the posterior end of the cavernous sinus to the upper knee of the lateral sinus, crossing the gasserian ganglion either from above or from below. Into the superior petrosal sinus drain the following veins.

1. The vein of the vestibular aqueduct.
2. Veins originating in the mucosa of the tympanic cavity and running through the petrosquamosal suture.
3. Veins originating in the periosteal layer of the bony capsule of the internal ear.
4. Veins anastomosing with the middle meningeal veins.
5. Occasionally the vein of the subarcuate fossa.
6. Occasionally a vein anastomosing with the inferior petrosal sinus.
7. Occasionally the vein of Labbé.
8. The petrosal vein, which carries the blood from the cerebellum.
9. The sinus ophthalmopetrosus (also called vein of Keck), which runs between the ophthalmic vein and the superior petrosal sinus or lateral sinus. It is often embedded in a fold of the dura. It originates in the outer angle of the superior orbital fissure and runs along the anterior surface of the petrous bone toward the petrosal sinus. In some instances the sinus consists simply of a vein embedded in the dura. It carries the blood from the ophthalmic vein to the sigmoid sinus.

Inferior petrosal sinus.—This sinus (Figs. 2 and 8) is usually larger than the superior petrosal. It arises from the posterior end of the cavernous sinus and passes back to the jugular foramen between the lower angle of the petrous bone and the adjacent border of the basilar portion of the occipital bone. It drains into the internal jugular vein or the inferior portion of the jugular bulb. The following veins drain into the inferior petrosal sinus.

1. The vein of the cochlear aqueduct, which is the principal vein of the internal ear.
2. Occasionally the vein of the subarcuate fossa.
3. Occasionally the vein of the vestibular aqueduct.
4. Frequently the internal auditory vein.
5. Branches from the basilar plexus.
6. Branches of the vertebral plexus running through the hypoglossal canal.

Sinus petrosquamosus.—To understand the anatomy of this sinus, a few embryologic findings must be recalled. In the early embryologic period, blood escapes from the skull through the primary head vein and the vertebral veins. Later the venous blood is carried through the sinus petrosquamosus which, at the zygomatic process of the temporal bone, passes through the skull and anastomoses with the external jugular vein. In the final embryologic phase, or in the first year of postfetal life, the sinus petrosquamosus becomes obliterated and the venous blood is carried through the lateral sinus and internal jugular vein. However, the sinus petrosquamosus sometimes persists either entirely or partially. In such cases there often are venules running from the middle cranial fossa toward the upper knee of the lateral sinus, or there is an actual sinus petrosquamosus originating from the upper knee. In these instances the sinus runs within the sulcus petrosquamosus of the tegmen tympani and leaves the skull through a foramen jugulare spurium, which may be found in the glenoid fossa of the mandible, the malar process of the temporal bone or the glaserian fissure. In some instances the sinus petrosquamosus drains into the middle meningeal veins, leaving the skull through the foramen spinosum. Several veins originating in the mucosa of the tympanic cavity drain into the sinus. These veins are particularly numerous in children.

Blood circulation.—Whereas in other organs expansion and contraction take place according to the phase of activity, the central nervous system is enclosed in a rigid box (skull and vertebral column) which does not allow volume changes of its contents. The Monro-Kellie doctrine emphasizes that the bony

containers protect the central nervous system from all external pressure and leave it subject only to internal pressures brought to it through the blood stream or created by the activities of its cells. The strongest argument in favor of this thesis is the fact that in animals it is possible to create negative pressures of extreme degrees within the cranial cavity, e.g., by intravenous injection of strongly hypertonic solutions. If the central nervous system were not contained in a rigid box, the walls of the container would yield to the negative pressure and collapse and so restore the positive pressure within the cranial cavity. However, certain observations do not entirely support the Monroe-Kellie doctrine. First, the skull of newborn and infants cannot be considered rigid, although negative pressures may occur in the cranial cavity of infants. Furthermore, with the skull intact and intracranial pressure within physiologic limits, the flow in the meningeal veins is steady, not pulsatile. Were the skull a rigid box, for every cubic centimeter of blood driven into the cranium, simultaneously with each heart beat an equal quantity would have to be driven out by way of the veins, because no increase in total volume within the rigid box would be possible. The flow throughout the system would be pulsatile. Likewise, a negative pressure, noted in the cranium when the upright position is assumed, could not occur if the central nervous system were enclosed in a truly rigid container.

For these and other reasons the bony containers of the central nervous system must be considered not as a strictly rigid box but as a chamber with rigid walls and expansile windows which are *plastic enough to permit physiologic variations* in the amount of cerebral blood. These modifications are brought about by the elastic membranes at the base of the skull and in the vertebral column, which yield to increased pressure; by the cranial nerves, which are somewhat movable at their exits; by the venous plexuses in the vertebral column, which are compressible, and by the cerebrospinal fluid, which can be displaced into the vertebral column. Despite these adjustments, the total contents of the cranium are, within physiologic limits, approximately equal.

Variations in any one of the three elements—brain, blood and cerebrospinal fluid—cause immediately reciprocal variations in one or both of the remaining elements.

The most variable element in the cranial cavity is the blood. It enters the brain through the large arteries. The normal arterial pressure is 1,500–1,600 mm. of water (120 mm. Hg) systolic and 1,100 mm. of water (80 mm. Hg) diastolic. Therefore the pulse pressure is approximately 500 mm. of water (40 mm. Hg). In the capillaries the pressure drops to 160 or 170 mm. of water, and the pressure in the veins averages 150 mm. of water (10 mm. Hg). This striking drop in pressure occurs chiefly in the arterioles. The arterial pulse is transmitted to the arterioles but is lost in the capillaries, in which the pressure is in closer relation to the venous than to the arterial pressure. Although a change of 500 mm. of water in arterial pressure does not cause a striking change in capillary and venous pressure, a variation of 10 mm. of water in venous pressure changes capillary pressure in the same direction to almost the same degree. The range between intracranial arterial and venous pressures regulates the rate of blood flow. Increase in arterial pressure causes chiefly an increased rate of blood flow and only a slight temporary rise in cerebral venous pressure.

Since the arterioles are the point of greatest resistance to blood flow, the degree of contraction or dilatation of these vessels regulates the intracranial arterial pressure as well as the capillary pressure, the two pressures being influenced in diametrically opposite directions. If the arterioles dilate, thus decreasing the resistance to blood flow, the arterial pressure must fall; but the capillary and venous pressure will rise, since the arterial pressure is now more directly transmitted to the capillaries and veins through the dilated arterioles. If the arterioles contract, thus increasing the resistance to blood flow, the arterial pressure will rise and the capillary and venous pressures will fall. The changes in the caliber of the arterioles depend not only on the systemic arterial pressure but also on the function of the vasomotor nerves, which constitute the intrinsic vasomotor mechanism of the

brain and are independent of the systemic arterial pressure.

Although changes in intracranial arterial pressure do not influence materially the venous intracranial pressure, the latter is dependent on the systemic venous pressure, postural changes, cerebrospinal fluid pressure and aspirating force of the right side of the heart and thorax. Among these factors, the systemic venous pressure is apparently of minor importance. Postural changes cause the same alteration in cerebral venous pressure as in cerebrospinal fluid pressure (p. 46); however, the magnitude of the first exceeds that of the second. Alterations in pressure of the cerebrospinal fluid cause, within physiologic limits, variations in the intracranial venous pressure of lesser extent but in the same direction. Under normal conditions the pressure in the cerebral and meningeal veins is practically the same as that of the cerebrospinal fluid. Otherwise the veins would be compressed by the cerebrospinal fluid pressure. The pressure of the cerebrospinal fluid is normally from 5 to 50 mm. of water above the pressure in the longitudinal sinus. In human beings the cerebrospinal fluid pressure is also higher than the pressure in the jugular vein. Nevertheless a compression of the sinuses is scarcely possible under normal circumstances, although the sinuses are compressible and distensible. However, compressibility is limited. First, the walls of the sinuses are comparatively thick, and a force of low degree will neither compress nor distend them. Furthermore, the sinuses are compressible only when they are not adherent to the bone. The deeper the sinuses are embedded in bone, the less compressible and distensible they are. Obviously the compressibility and distensibility of the sinuses are increased if the bone to which the sinus is adherent is removed.

Of great importance is the aspirating force of the chest. This force acts especially on those venous channels which are closest to the heart, namely, the jugular vein and the sigmoid sinus. If the variations in intrathoracic pressure were to act to the full extent on the sigmoid sinus, there would be a considerable disturbance in the cerebral blood circulation and in the normal

activity of the brain. Therefore the effect of the aspirating force on the sigmoid sinus must be attenuated. This purpose is achieved by the curved course of the sigmoid sinus, the narrow osseous dimension of the exit of the jugular fossa, the level of the outlet of the jugular fossa, which is obliquely forward while the course of the jugular vein beyond is obliquely downward, and the shape of the jugular bulb itself. All of these anatomic characteristics are variable, particularly the shape of the jugular bulb. If the jugular bulb forms a high dome, only a small amount of the aspirating force will act on the sinus, the greater part being lost in the jugular bulb. Consequently, if the jugular bulb is shallow, a greater amount of the aspirating force will influence the sinus. However, since the shape of the jugular bulb is not the sole protection for the sigmoid sinus against the aspirating force of the chest, the negative pressure in the sigmoid sinus during inspiration is attenuated in all circumstances. For this reason, air embolism following injuries of the sinus is extremely rare.

PIA-ARACHNOID (LEPTOMENING)

The pia-arachnoid is a membrane consisting chiefly of connective tissue. The separation of pia and arachnoid is artificial. Nevertheless, from a practical point of view, the separation is useful.

The arachnoid has two layers. The external layer consists of delicate connective tissue, is covered by a low mesothelium and does not contain nerves or blood vessels. The internal layer is connected intermittently with the pia. Most of the pia-arachnoid is not sensitive to pain. Between the two layers of the arachnoid is a network of connective tissue which frequently forms fine membranes covered by mesothelium. Occasionally several trabeculae of connective tissue grow together and form cystlike cavities. Numerous blood vessels run between the connective tissue strands and membranes. These blood vessels, which seldom have a distinct adventitia, are suspended by the fibers of the network or are adjacent and adherent to the membranes.

The external layer frequently presents cell clusters, probably

owing to a proliferation of the mesothelial cells of the arachnoid. Later the cell clusters may show various types of degeneration. Often at the site of, or close to, the cell clusters are found arachnoid villi, called pacchionian bodies if they reach a considerable size.

Pacchionian bodies.—At certain sites villi rise from the external layer of the arachnoid. They consist of a loose connective tissue which is covered by the mesothelium of the arachnoid and contains calcareous bodies and, rarely, capillaries (Fig. 11). The arachnoid villi, although of microscopic size, are present

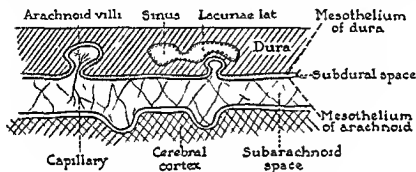


FIG. 11.—Arachnoid villi.

even in infants. Gradually they enlarge to macroscopic formations which rarely are noted prior to the third year but usually are found after the tenth year. They extend into the subdural space and later invade the dura. They may cause the walls of the lateral lacunae of the sinuses to bulge into the lumen and make contact with the blood of the sinuses. At the convexity of the brain the arachnoid villi often form a network. The tufts may become so large that the bones of the skull are eroded either by the villi or by the lacunae of the sinuses.

Under certain circumstances the cerebral cortex may perforate the pia and extend into a pacchionian body. In this event the pacchionian body is transformed into a formation called hernia of the cerebral substance. These minute cerebral protrusions are especially likely to be found at the base of the skull. Figure 12 shows the areas at the base of the skull which

often present both cell clusters of the arachnoid and cerebral herniations. Cerebral herniations are noted especially in instances of long-standing increased brain pressure, although they have been discovered at the base of the skull when autopsy did not reveal any cause of increased pressure. Some writers attribute these "physiologic herniations of the brain" to a congenital leptomeningeal defect and others to transitory periods of increased intracranial pressure.

Figure 12 indicates that the arachnoid cell clusters occur particularly at the cribriform plate, in the area of the eustachian tube and the tegmen tympani and occasionally on the posterior surface of the petrous bone and close to the lateral sinus. In the temporal bone, there is microscopic evidence that at the same sites where the cell clusters are found, cerebral herniations frequently occur. That is particularly true for the tegmen tympani. There are cases on record in which cerebral herniations bulged deep into the tympanic cavity, filling a large part of the epitympanum and being covered by a thickened mucous membrane of the tympanic cavity. In the cribriform plate, whether cerebral herniations and arachnoid villi are as common as arachnoid cell clusters remains to be shown. In some instances, idiopathic cerebrospinal rhinorrhea and liquorrhea subsequent to intranasal surgery may perhaps be due to rupture of arachnoid villi in the area of the cribriform plate.

Cephaloceles.—Although in the ear the arachnoid villi and cerebral herniations are, as a rule, of minute size and apparently do not occur in infants, they occasionally attain the size of a tumor in the nasal cavity. These tumors, which are congenital in origin, are called cephaloceles. They bulge into the nasal cavity or the epipharynx and consist of protruded meninges only, of brain substance only or of brain substance plus meninges. They may or may not communicate with the dilated anterior horn of the lateral ventricle, with the third ventricle or with the rudimentary ventricle of the olfactory bulb. Embryologic investigations have proved that cephaloceles are the primary formations which are surrounded secondarily by bone at their entrance into

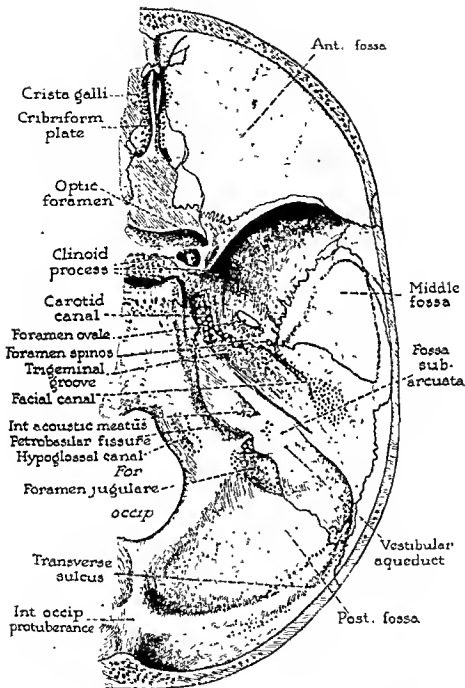


FIG. 12.—In the dotted areas, arachnoid cell clusters and herniations of the brain are frequently noted. (After Aoyagi and Kyuno, from Cushing, Brain 45:282, 1922, completed by the author.)

the nasal cavity or the epipharynx. According to their contents, cephaloceles are called encephaloceles, encephalocystoceles, encephalocystomeningoceles or meningoceles. Other congenital deformities of the skull or brain are frequently associated with cephaloceles. From the point of view of the otolaryngologist, the distinction between intranasal and extranasal cephaloceles is important.

Among extranasal cephaloceles, the following types are recognized. (1) The nasofrontal cephalocele, which protrudes between the frontal and nasal bones, lies on the outside of the nasal bones and has no communication with the inside of the nose. (2) The naso-ethmoid cephalocele, which protrudes through the foramen caecum, extends beneath the nasal bones and is separated from the inside of the nose by the ethmoid process of the frontal bone. (3) The naso-orbital cephalocele, which protrudes through the most anterior part of the cribriform plate and through a defect of the mesial wall of the orbit, pushes the lateral nasal wall toward the nasal septum and enters the orbit.

Among the intranasal cephaloceles, the following types are recognized (Fig. 13). (1) The transethmoid cephalocele protrudes through a defect in the cribriform plate and bulges into the superior nasal meatus, simulating a nasal polyp. (2) The spheno-ethmoid cephalocele protrudes between ethmoid and sphenoid and extends particularly into the epipharynx. (3) The trans-sphenoid cephalocele protrudes through a patent cranio-pharyngeal canal into the pharynx, occasionally simulating adenoids.

Among the cephaloceles, the transethmoid are most common and have most practical importance. The patients frequently present a widening of the bridge of the nose and an increase of distance between the eyes, and they complain of headache, blocked nose and persistent liquorrhea after rupture of the cephalocele. Rhinoscopic examination reveals what seems to be a nasal polyp in the superior or the middle nasal meatus lateral to the anterior end of the middle turbinate. The cephalocele may

push the nasal septum to the other side and may be attached to the nasal mucosa by adhesions. Since the cephalocele almost never shows pulsating movements, proper diagnosis presents considerable difficulty. In infants nasal polyps are always suggestive



FIG. 13.—Sites of cephaloceles: 1, naso-ethmoid, 2, transethmoid, 3, sphenothmoid; 4, trans-sphenoid. a, cribriform plate; b, frontal sinus; c, sphenoid sinus; d, optic foramen; e, superior orbital fissure; f, vidian canal; g, pterygoid process; h, foramen ovale; i, foramen lacerum; k, ethmoid; l, foramen spinosum; A, insertion of pharyngeal wall at base of skull.

of foreign bodies, malignant tumors or cephaloceles. Simple nasal polyps are rare in infants. In adults, liquorrhea may facilitate the diagnosis. If the correct diagnosis is made by means of clinical and x-ray examination, plastic closure of the defect in the cribriform plate should be given a trial.

Pia mater.—This is a membrane consisting of connective tis-

sue which is inseparably grown together with the internal layer of the arachnoid. The pia contains numerous blood vessels and nerves. The blood vessels anastomose with the blood vessels of the brain (p. 81) and with the internal network of the dura (p. 3). Where an anastomosing blood vessel originating from

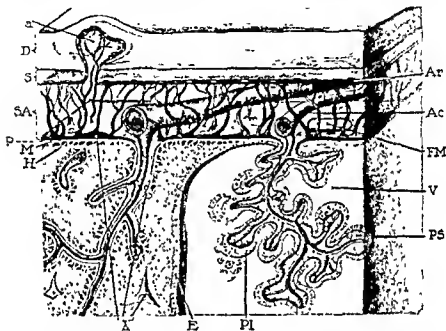


FIG 14—External and internal cerebrospinal fluid spaces. *a*, pachionian bodies, *D*, dura, *S*, subdural space; *SA*, subarachnoid space, *P*, pia, *M*, membrana gliae limitans, *H*, glia layer of Held, *A*, artefact, *E*, ependyma, *PI*, epithelium of choroid plexus, *Ar*, arachnoid, *Ac*, choroid artery, *FM*, foramen of Magendie, *V*, ventricle, *PS*, stroma of choroid plexus (Adapted from Spatz)

the veins of the pia approaches the dura, the arachnoid bulges toward the dura and forms a sheath around the blood vessel. Where the blood vessel enters the dura, the arachnoid fuses with the connective tissue of the dura, and the mesothelium of the arachnoid joins the mesothelium of the dura. The nerves of the pia either run together with the blood vessels or are independent of the blood vessels. They arise from the sympathetic nerves accompanying the carotid and vertebral arteries and from cranial nerves.

INTERMENINGEAL SPACES

Between dura and arachnoid is a potential space, the *subdural space*, bounded on both sides by mesothelium (Fig. 14). The space is crossed by blood vessels, nerves, arachnoid villi and

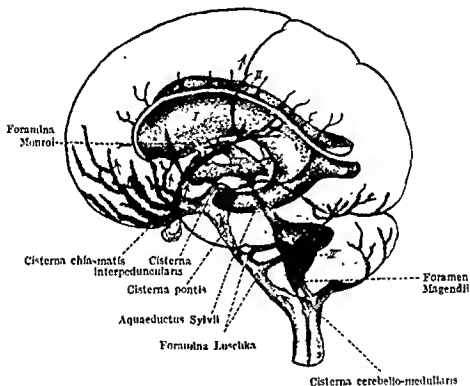


FIG. 15.—Ventricles of the brain and the subarachnoid spaces. Arrows indicate the movement of cerebrospinal fluid. I and II, lateral ventricles, III, third ventricle; IV, fourth ventricle. (After Dandy.)

the infundibulum of the third ventricle, which penetrates the diaphragm of the sella turcica. There is no open communication between the subdural space and the subarachnoid space or between the subdural space and the ventricles. The subdural space has no efferent vessels and contains only a small amount of fluid. If the dura is incised without injury to the arachnoid, only a few drops of fluid escape under normal circumstances.

Between the two layers of the arachnoid is the *subarachnoid*

space (Fig. 14). Especially at the base of the brain, this space forms large cavities, called the *arachnoid cisternae*, which exhibit considerable variability of their extension. They constitute a fluid cushion between brain and bone. In fact, the only parts of the base of the brain which rest directly on bone are those in contact with the orbital plates of the frontal bone and those impinging on the lesser wings of the sphenoid. The following cisternae are important to the otolaryngologist (Fig. 15).

1. Cisterna cerebellomedullaris, or cisterna magna. This communicates with the subarachnoid space of the spinal cord backward and with the cisterna pontis forward. It is bounded by the external layer of the pia-arachnoid, which extends from the dorsal surface of the medulla oblongata to the posterior surface of the cerebellum. The cisterna is adjacent forward to the medulla oblongata and backward to the dura beneath the atlanto-occipital membrane (Fig. 20, p. 64). The diameter of the cisterna varies considerably. The maximal depth varies between 1.5 and 2 cm. and the maximal width between 5 and 6 cm. As a rule, the cisterna consists of one single space which communicates with the fourth ventricle through the foramen of Magendie and foramina of Luschka; occasionally the space is crossed by strands of connective tissue. The cisterna magna can be tapped by occipital puncture (Fig. 20).

2. Cisterna pontis (Fig. 16, see also Fig. 61, p. 284). This is frequently divided into a cisterna pontis media and two cisternae pontis laterales, although the separation is rarely distinct. The cisterna contains the basilar artery and trigeminal, abducens, facial, acoustic, glossopharyngeal and vagus nerves and communicates with the cisternae in front of the pons. The cisterna pontis lateralis is close to the petrous bone, extending from the apex toward the posterior surface of the petrous bone, where the internal auditory meatus and cochlear aqueduct drain into the cisterna.

It is possible to reach the cisterna from the mastoid process if the dura is carefully lifted from the posterior surface of the petrous bone. If this procedure is performed 3-5 mm. below the

superior angle of the petrous bone, one can tap the cisterna without injuring the labyrinth or the endolymphatic sac. There are cases on record in which a cisterna pontis lateralis extended close to the lateral sinus. In such instances an incision of the

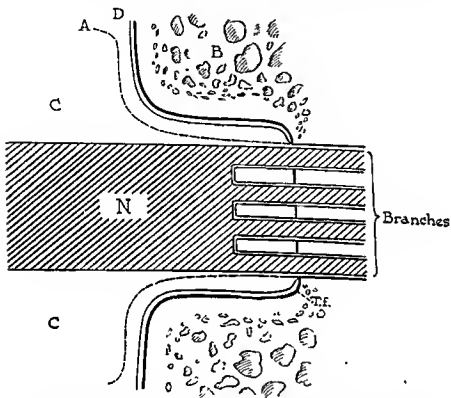


FIG. 16.—Cochlear and vestibular nerves (*N*) in the auditory meatus. *C*, cisterna pontis; *A*, arachnoid, *D*, dura; *B*, petrous bone; *Tf*, tractus foraminosus.

dura of the posterior fossa mesial to the lateral sinus might strike the cisterna, causing a flow of cerebrospinal fluid. Occasionally the dura in the angle between the lateral sinus and the superior petrosal sinus is bulged toward the mastoid cavity owing to an enlarged cisterna. Since the dura is always thin at that point, injury of the dura and the cisterna beneath it may occur in the course of a mastoid operation.

3. Cisterna of the gasserian ganglion. This is located within Meckel's cavity (Fig. 2). As was mentioned previously, the root of the trigeminal nerve and the gasserian ganglion occupy a

diverticulum of the dura of the posterior cranial fossa. Both the root of the nerve and the concave surface of the ganglion are covered by one or two layers of flat cells which provide a sheath for the nerve and originate from the pia-arachnoid of the brain. Numerous spotlike accumulations of arachnoid cells and calcareous bodies, frequently noted in the cerebral arachnoid, accompany the root of the trigeminal nerve in adults. Between the bundles of the root is ample space, particularly within the concavity of the ganglion, filled with cerebrospinal fluid. Since the space communicates along the root of the trigeminal with the pontile cisternae, it must be considered an arachnoid cisterna within the cavity of Meckel. For this reason, an infection in the cavity of Meckel must be looked upon as localized meningitis (Fig. 62, p. 285).

4. Cisterna of the chiasma. This is located beneath and in front of the optic chiasma in the angle formed by the divergent optic nerves. In pathologic circumstances the cisterna may be distended and exert strong pressure on the optic nerves. The cisterna communicates forward with the cisterna laminae cinereae terminalis, which extends upward to the corpus callosum. In instances of a very large sphenoid sinus there is only a thin layer of bone between the cisterna and the sphenoid sinus.

The absolute capacity of the intermeningeal spaces and also their capacity in relation to that of the skull is considerably smaller in the newborn than in adults. In the newborn the brain occupies 97½ per cent of the volume of the skull and the intermeningeal spaces only 2½ per cent. At the end of puberty the brain occupies only 92½ per cent of the volume of the skull and 7½ per cent of the skull volume is available for the intermeningeal spaces. For this reason, only a slight swelling of the infant brain will push the brain toward the bones of the skull and cause cerebral symptoms.

Both the subarachnoid and the subdural space extend into the internal ear and nasal cavity and, indirectly, into the brain. In that respect the following anatomic findings are important.

1. Cochlear aqueduct. In both children and adults the inter-

meningeal spaces extend deep into the cochlear aqueduct. The extension of both spaces within the aqueduct varies from case to case; frequently the connective tissue of the subarachnoid space extends up to the scala tympani of the vestibular portion of the cochlea where it blends with the periosteum of the scala. Although from the anatomic point of view the cochlear aqueduct furnishes a free communication between the perilymph of the internal ear and the cerebrospinal fluid of the subarachnoid

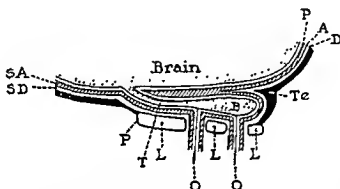


FIG. 17.—Sagittal section through olfactory bulb (B) and cribriform plate (L). P, pia; A, arachnoid; D, dura; T, olfactory tract; SA, subarachnoid space; SD, subdural space; P, periosteum of cribriform plate; O, olfactory fibers; Te, tentorium of olfactory bulb.

space, in adults the channel is apparently too narrow to encourage a liberal mingling of the two fluids. However, the channel does permit invasion of bacteria and leukocytes from the subarachnoid space into the internal ear, as occurs in epidemic cerebrospinal meningitis.

2. *Internal auditory meatus* (Fig. 16). The intermeningeal spaces extend into the internal auditory meatus. The auditory nerve within the meatus is surrounded by an arachnoid sheath which, at the fundus of the meatus, blends with the dural periosteum of the channel. Where the nerve enters the cavity of the skull the arachnoid sheath blends with the external layer of the arachnoid of the cisterna pontis, and the nerve runs through the cisterna pontis toward the medulla oblongata.

3. *Lamina cribrosa*. The intermeningeal spaces accompany-

ing the bundles of the olfactory nerve extend into the olfactory membrane of the nasal cavity (Fig. 17). The current of spinal fluid along the olfactory filaments is thought to be centrifugal. Next to the meningeal sheaths of the olfactory nerve is a network of lymph capillaries in the olfactory area of the nose which, in the newborn and infants, is believed to be in communication with the subarachnoid space. The communication is provided by small canaliculi which pass through the foramina of the cribriform plate with the filaments of the olfactory nerve. The network of lymph capillaries has no communication with the lymphatics of the respiratory part of the nose, because the latter run back toward the epipharynx and drain into the lymph nodes of the pharynx and neck. Whether the lymph-capillary network of the olfactory area in adults communicates with the subarachnoid space is questioned by many investigators. I have noted, in adults, lymphatics running in the mucosa of the lateral wall of the nose from the olfactory area up to the cribriform plate. However, it was not possible to trace these lymphatics to the subarachnoid space.

4. Cerebral blood vessels. The cerebral blood vessels pierce the dura and arachnoid and extend to the pia (Fig. 14). If they extend into the brain they force the pia ahead, the pia forming a funnel-like opening toward the subarachnoid space. Along the blood vessels the pia is firmly attached to the superficial glia of the brain, called the *membrana limitans gliae*. There is no space between the pia and the superficial glia. However, between the pia and the wall of the blood vessel in normal circumstances there is a potential space, called the space of Virchow-Robin. With the decrease in caliber of the blood vessel the width of the space of Virchow-Robin decreases, and around the capillaries the space is poorly developed or absent. Within the space of Virchow-Robin is a loose network of connective tissue anastomosing with the connective tissue of the pia-arachnoid.

It is assumed that in the area of the cerebral blood vessels there is a hemato-encephalic barrier which permits certain contents of the blood to pass into the brain while other contents are

retained. Blood contents which pass it are aniline dyes, toxins, viruses, antibodies and drugs, e.g., sulfonamides and urotropin; contents which are retained include tetanus and diphtheria toxin and penicillin. There is no unanimity concerning the exact localization and function of the barrier. Nor is it definitely determined that this is the only barrier in the brain. Some investigators claim that there is not only a barrier between blood and cerebrospinal fluid but likewise one between cerebrospinal fluid and brain. Still others do not consider the term "barrier" entirely adequate because the problem is only a part of the more general problem of capillary permeability. To the otolaryngologist it is noteworthy that in the newborn and infants the barrier between blood and brain is more permeable than it is in adults and that acute infections of the meninges render the barrier more permeable. The latter fact encourages conservatism in treatment of meningitis and also explains why, in patients with a positive Wassermann reaction of the blood, the Wassermann reaction of the cerebrospinal fluid may become positive if there is meningitis.

CEREBROSPINAL FLUID

Present knowledge of the processes of the cerebrospinal fluid is inadequate in many respects, particularly regarding strictly physiologic conditions in man. A great deal of work has been done on animals. However, because the dynamics in the skull differ in man and the other mammals and because the preparation of the experiment usually creates circumstances which alter the strictly physiologic conditions, it is only with great caution that conclusions drawn from animal experiments can be applied to man.

Sources of cerebrospinal fluid.—The conception that cerebrospinal fluid is produced principally by the intraventricular choroid plexuses is based not on any single conclusive piece of evidence but on a number of suggestive data. When considered from all standpoints, the hypothesis seems well established. Whether the ependyma of the ventricles, the blood vessels of the meninges and the brain tissue share in the production of

the human cerebrospinal fluid under normal circumstances cannot be stated. The evidence indicates that there is a constant formation of cerebrospinal fluid under physiologic conditions. The total amount in the cerebral ventricles and around the nervous system in adults is 100–150 cc. Accepting the premise that the choroid plexuses are the principal elaborators of cerebrospinal fluid, the question arises whether the fluid is a true secretion or a dialysate. The present, although by no means unanimous, opinion seems to favor the dialysate theory. In that case the rate of formation will be encouraged by the capillary pressure in the choroid plexus. Opposing this is the osmotic pressure of the plasma proteins to which the capillary wall is impermeable. Under physiologic conditions the protein content of the blood, and thus the osmotic pressure, does not show appreciable changes. For this reason, the rate of formation of cerebrospinal fluid depends largely on capillary pressure, particularly in its venous portion. The pressure at which the fluid comes through the choroid plexus will be equal to the capillary pressure minus osmotic pressure of the plasma proteins. Under most conditions this pressure will vary directly with the pressure in the venous portion of the capillaries of the choroid plexus.

Absorption of cerebrospinal fluid.—It appears that absorption of cerebrospinal fluid is a twofold process, being chiefly one of rapid drainage into the dural sinuses and, to a slight extent, one of slow indirect escape into the true lymphatic vessels. Whether the veins of the pia, the cortical cerebral vessels and the ependyma of the ventricles share in the absorption of cerebrospinal fluid is not certain, at least in adults and under normal conditions.

The chief pathways along which the cerebrospinal fluid is absorbed are (1) the arachnoid villi and pacchionian bodies (p. 31), (2) the lymphatics and (3) the nerve sheaths. As pathways for the outflow of cerebrospinal fluid, the lymphatics are more important in animals than in man, and in both animals and man they are more important for drainage of the spinal sac than for drainage of the subarachnoid spaces of the brain. For drainage of the subarachnoid spaces of the human

brain, the lymphatics in the upper part of the nose might be considered provided they actually exist (p. 42). The third pathway is furnished by the sheaths of all cranial and spinal nerves, particularly those of the olfactory filaments, optic nerve and acoustic nerve. These pathways carry the fluid primarily in a centrifugal direction. Some writers claim that the pathways carry it in a centripetal as well as a centrifugal direction, particularly so far as the sheaths of the optic nerve are concerned. The total effective force which determines absorption, under normal circumstances, apparently is compounded of hydrostatic pressure (subarachnoid pressure minus cerebral venous pressure) and osmotic pressure of the blood.

Circulation of cerebrospinal fluid.—The cerebrospinal fluid is poured directly into the cerebral ventricles. That formed in the lateral ventricles flows through the foramen of Monro into the third ventricle and thence through the sylvian aqueduct into the fourth ventricle. From the fourth ventricle the fluid passes into the cisterna magna and cisterna pontis through the foramen of Magendie and foramina of Luschka. From the great cisternae the fluid slowly seeps down into the spinal subarachnoid space, but passes more rapidly upward about the base of the brain and then more slowly over the hemispheres, where it is absorbed in the sinuses and veins of the meninges (Fig. 15). The circulation of cerebrospinal fluid, normally very sluggish within the skull, is actuated by impulses transmitted by the pressure of the secretion, by the position of the body, by respiration, by vascular pulsations and by movements of the head and body. Hydrostatic forces appear to take no significant, if any, part in the displacement of ventricular fluid into the subarachnoid spaces. A flow of cerebrospinal fluid from the ventricles through the brain substance into the subarachnoid spaces has frequently been assumed, but it probably does not exist, at least under normal conditions. There are many controversial questions concerning the circulation of the cerebrospinal fluid. Some neurologists even doubt that there is any circulation of cerebrospinal fluid.

Increase of fluid leads to hydrocephalus, which may be

caused (1) by occlusion of the arachnoid villi, (2) more rarely by an increase in the amount of fluid secreted in excess of what can be cared for by the normal portals of drainage, and (3) by obstruction, notably at the foramina of Monro, in the sylvian aqueduct, in the fourth ventricle or in the area of the foramen of Magendie and foramina of Luschka. Obstructions of the channels of the cerebrospinal fluid are commonly caused by otorhinogenous brain abscesses.

Pressure of cerebrospinal fluid.—Normal pressure is determined largely by the balance between the constant production of cerebrospinal fluid in the cerebral ventricles and its absorption into the dural sinuses. Changes of pressure, within physiologic limits, are caused by changes of position of the body, osmotic pressure of the blood, respiration and, to a certain extent, by the intracranial arterial and venous pressures. When the body is in the lateral recumbent position, the pressure of cerebrospinal fluid is approximately the same in all parts of the craniovertebral cavity. It rises from 80 to 150 mm. in adults and from 50 to 100 mm. in children. Several authors have noted a slight increase of pressure in the ventricles, which they called "secretion pressure of cerebrospinal fluid," and others have found a slight increase of pressure in the lumbar portion of the cord. In neither instance is the increase of practical importance. With change from the horizontal to the erect position the hydrostatic pressure of the fluid comes into action. In that event there is a negative pressure (less than atmospheric) in the skull. Although there is no consensus concerning the exact amount of pressure, one may assume that when the body is erect the pressure in the ventricles amounts to -150 up to -300 mm. of water and in the cisterna magna, -40 up to -70 mm.; at the level of the inferior portion of the cervical spine the fluid pressure reaches atmospheric pressure and gradually rises toward the lumbar portion of the spine. Here the pressure is between 250 and 350 mm. in adults in sitting position and in children, between 150 and 250 mm. All of these figures are approximations.

The difference in pressure in the erect and in the horizontal

position is due to the hydrostatic pressure of the fluid above the site of puncture. The amount of hydrostatic pressure is by no means constant; it is strikingly modified by the coefficient of elasticity of the spinal dural sac and the soft parts at the base of the skull. The pressure of the fluid column must decrease corresponding to the degree to which the spinal sac and the elastic tissue at the base of the skull yield to the pressure of the fluid. An effort was made to analyze the degree to which hydrostatic pressure is caused by the pressure of the fluid column and by the elasticity of the coverings, but no practical method was worked out.

Regarding osmotic pressure, the cerebrospinal fluid is in osmotic equilibrium with the plasma of the blood. The total osmotic pressure of plasma is about 5,400 mm. Hg; that is, if a membrane were to separate plasma from pure water and were impermeable to everything in the plasma except water, hydrostatic pressure equivalent to a column of mercury more than 5 m. high would have to be exerted on the plasma to prevent the passage of water across the membrane into it. However, the capillary wall is permeable to nearly all osmotically active substances in the blood except proteins. For this reason, only the osmotic pressure due to the plasma proteins must be considered, since all other substances depress the tendency of water molecules to escape across the membrane equally in both directions,¹ while the proteins encourage the passage of water into the blood.

Although osmotic pressure prevents passage of fluid across the capillary wall, capillary hydrostatic pressure tends to increase the escape of fluid into the tissue spaces. Normally these pressures are balanced: fluid filters into the tissue spaces from the arterial portion of the capillary bed, where hydrostatic pressure is higher than osmotic pressure, and is reabsorbed into the venous portion of the capillary bed, where osmotic pressure is higher than hydrostatic pressure. This normal fluid exchange requires an osmotic and hydrostatic equilibrium between plasma and cerebrospinal fluid. With hydrostatic pressure constant, vari-

¹This statement does not take into consideration the so-called Donnan effect.

ations in the relative osmotic pressure of blood and cerebrospinal fluid cause volume changes like edema or dehydration in tissues not rigidly confined; when they occur in the cranium the volume changes of the brain are limited, but changes in intracranial pressure do result. Intravenous injections of strongly hypertonic solutions, such as a 20-50 per cent glucose solution or a 10-20 per cent solution of sodium chloride, cause a definite fall of cerebrospinal fluid pressure and shrinking of the brain, whereas injections of iso- or hypotonic solutions cause an increase of pressure and swelling of the brain. The depressing effect of hypertonic solutions has practical significance, although it is temporary.

Much work has been done in analyzing the influence of drugs on the pressure of cerebrospinal fluid, but no definite information has been gained. Thyroid extract seems to have some value in certain instances of increased intracranial pressure, causing a moderate fall in pressure.

Respiration causes fluctuations in pressure of cerebrospinal fluid without influencing the permanent pressure. During lumbar puncture, the excursions of the column of cerebrospinal fluid in a standpipe manometer with each respiration show a variation in fluid pressure of 5-20 mm. of water. These variations in fluid pressure depend on the variations in venous pressure in the dural sinuses and epidural venous plexus of the spinal cord caused by respiration.

Since the brain is enclosed in an almost rigid box, volume changes in arteries or veins influence intracranial volume, and hence cerebrospinal fluid pressure, directly. The dependence of cerebrospinal fluid pressure on arterial pressure is not striking. Although pulse pressure in the larger arteries is about 500 mm. of water and in the circle of Willis at least 340 mm. of water, cerebrospinal fluid pressure rises only 2-6 mm. of water with each heart beat.

Otologists are familiar with the pulsating sinus, i.e., pulsating motions of the surgically exposed sinus. Likewise, in the presence of a perisinus abscess the pus often escapes in pulsating rhythm

during operation on the mastoid. There is no satisfactory explanation of these observations. In exceptional instances the negative pulse wave originating in the atrium of the right heart is transferred through the jugular vein into the sigmoid sinus. In most cases the blood in the sinus does not move with pulsating motions. If a pulsating sinus is incised, the blood squirts out in a continuous jet. Furthermore, pulsating motions of the sinus have been noted in the presence of an obliterating sinus thrombosis. For these reasons, the pulsations of the exposed sinus have been explained by a shifting of the sinus with the neighboring dura, caused by the arterial pulse in the cerebrospinal fluid. I doubt that a pulse pressure of 2-6 mm. of water is capable of causing a shifting of sinus and dura. However, it is conceivable that a shift of this type might be accomplished if there were active hyperemia of the leptomeninges beneath the sinus and dura, caused by serous meningitis. According to this concept, a pulsating sinus would indicate serous meningitis in the posterior cranial fossa.

Further proof of the relative independence of arterial and cerebrospinal fluid pressure is the observation that arterial hypertension has no constant influence on the pressure of the cerebrospinal fluid unless there is a renal complication, disturbing the osmotic equilibrium between plasma and cerebrospinal fluid.

Although variations in arterial pressure do not materially influence intracranial pressure, the latter follows venous pressure changes almost quantitatively. To cause a rise in pressure of the cerebrospinal fluid, the increase in venous pressure must occur rapidly. If the obstruction to outflow of venous blood develops slowly and persists for a long period, as in sinus thrombosis, *there is no appreciable rise in intracranial pressure. But if the jugular vein is compressed suddenly, there is a prompt, adequate rise in pressure of the cerebrospinal fluid.* These facts are the basis of the Queckenstedt test, which is performed in the following manner.

Pressure is exerted on one or both jugular veins, which causes a rise in pressure of the cerebrospinal fluid. Normally, the increase in pressure spreads rapidly in the lumbar cord, causing a rise of the fluid

in the manometer less than one second after the compression that continues rapidly to the maximal position. With diseases causing an obstruction in the spinal cord, the rise in the manometer is markedly delayed and after release of the compression of the vein the pressure of the cerebrospinal fluid returns to normal very slowly or not at all.

Tobey and Ayer applied the Queckenstedt test in the diagnosis of lateral sinus thrombosis, using the following technic.

With the patient in the lateral position and the suspected sinus lowermost, a lumbar puncture is performed and fluid is allowed to run into a glass manometer of 2 mm. bore. If, for some reason, the ventricles are punctured, fluid pressure in the ventricles can be measured. The initial pressure reading is noted. Pressure is then exerted on one side of the neck between the larynx and the sternocleidomastoid muscle until a strong carotid pulse is felt. The vessel is pressed against the transverse process of the cervical vertebra, not against the larynx, with care not to compress the external jugular vein. During the compression, the surgeon watches the rapidity of rise of the fluid column in the manometer, the promptness of its beginning, the final height it attains and, on release of jugular compression, the rapidity of drop in pressure. The procedure is then repeated on the opposite side of the neck. Since cerebrospinal fluid pressure is somewhat diminished in the vein on the dependent side, readings should be taken from the upper side by reversing the patient's position. Finally, for comparison, both jugular veins are compressed simultaneously.

In a typical case of lateral sinus thrombosis there is a prompt and rapid rise in fluid pressure, to twice or three times the initial reading, on compression of the jugular vein on the normal side. This pressure rise is maximal, being equivalent to the pressure attained when both jugular veins are compressed. Pressure over the vein draining the thrombosed lateral sinus causes either no rise or, more commonly, a slow rise of only 10-20 mm. in the manometer. Partial obstruction by a mural thrombus gives less striking results. For diagnosis of lateral sinus thrombosis, the difference in rise on the two sides must be striking, because in normal individuals there is often a considerable difference in the height of pressure reading following compression of each jugular vein separately. However, in normal subjects a difference of over 50 mm. is unusual and over 100 mm., exceptional.

The Tobey-Ayer test is believed to facilitate the diagnosis of unilateral sinus thrombosis in cases of bilateral otitis, which is

common in children. But frequently it does not succeed. Jugular compression on the involved side may fail to show a rise in cerebrospinal fluid pressure even though there is no thrombosis of the lateral sinus, and vice versa. The response of cerebrospinal fluid to jugular compression depends largely on (1) the technic of compressing the veins, (2) initial pressure of the cerebrospinal fluid, which should not be over 200 mm., (3) leakage of spinal fluid through the puncture, (4) hyperventilation, (5) variations in intracranial blood flow, (6) thick neck, (7) anatomic variations of the sinuses and (8) interfering movements, such as coughing and movements of the head. The multitude of influencing factors explains the frequency of failures. Nevertheless the test should always be performed if there are indications for spinal puncture. I have never been compelled to perform a spinal puncture for the sake of the Tobey-Ayer test. Thorough examination of the ears usually reveals, even in children with bilateral otitis, that the otitis is more advanced on one side than on the other. Sinus phlebitis is usually found on the side where the otitis is more advanced. X-ray examination of the mastoids offers further support in these instances.

Intracranial hypertension.—Intracranial hypertension occurs if the normal relationship between the available capacity of the skull and the contents of the skull is disturbed. Each of the contents of the skull—cerebrospinal fluid, blood and brain—is capable, under pathologic conditions, of disturbing this relationship.

1. Cerebrospinal fluid will cause intracranial hypertension (a) if there is excessive production of fluid, (b) if there is delayed absorption of fluid, (c) if there is an obstruction of the circulation of fluid and (d) if all of these factors, or some of them, are acting. Increased pressure of the cerebrospinal fluid is transmitted readily to the interior of the thin-walled veins. Thus the intracranial venous pressure is raised and the blood flow retarded. The rise in venous pressure is transmitted to the capillaries, then to the arterioles and small arteries, raising the pressure in each and causing dilatation of all these vessels. Since the constant arterial pressure is about six times the venous pressure, venous

and capillary pressure can rise considerably without approaching that of the larger arteries. For this reason, the constant arterial pressure establishes a slower, yet effective, circulation in the brain without causing a rise in systemic arterial pressure.

It is the compensatory effect of cerebral arterial pressure in the presence of intracranial hypertension which prevents intracranial pressure raised to a level approaching cerebral arterial pressure, at least diastolic pressure, from shutting off cerebral circulation. For this reason, patients with general intracranial pressure of gradual development and short duration may have a functioning cerebral circulation with normal arterial pressure, normal pulse and normal respiration. Only the pulsations of the brain are increased, and there may be passive hyperemia of the retinal veins or even beginning papilledema.

When intracranial hypertension reaches or exceeds the level of arterial pressure, cerebral anemia with blanching of the cerebral cortex occurs. Cushing demonstrated the existence of a hind-brain regulatory mechanism. This mechanism acts on a partial anemia of the vasomotor center in the hindbrain and brings about a rise in systemic blood pressure great enough to maintain cerebral circulation during excessive elevations of intracranial pressure. This rise in systemic arterial pressure is called the blood pressure reflex of Cushing. It occurs only when the rise in intracranial pressure is sudden and marked, as in intracranial injury and terminal phases of expanding lesions. With intracranial complications it is rare.

All of these mechanisms of compensation except the blood pressure reflex of Cushing are active when the increase in intracranial pressure is gradual. If, however, the intracranial pressure is raised rapidly and to the level of the systemic blood pressure, respiration may cease immediately.

2. The cerebral arteries as well as the cerebral veins may cause intracranial hypertension under pathologic conditions. Obstruction of the arterial lumen and rupture of the arterial wall are followed by an increase in intracranial pressure. However, these conditions do not concern the otolaryngologist. More im-

portant factors in causation of intracranial hypertension are the cerebral veins and dural sinuses. Compression of the jugular veins in normal circumstances causes a rise in cerebrospinal fluid pressure. Since the rise in pressure ceases immediately after release of the compression, it can hardly be caused by excessive production or delayed absorption and in all probability is caused by the displacement of cerebrospinal fluid by venous stasis in the cranial cavity. In sinus thrombosis the outflow of venous blood is also impeded, but this impediment develops slowly, in contrast with that following compression of the jugular vein. Consequently, in sinus thrombosis an increase in intracranial pressure is not to be expected. Nevertheless, practical experience proves that in 10-16 per cent of cases papilledema occurs. In some, papilledema appears after ligation of the jugular vein, in others prior to it. In the latter cases the impediment to outflow of venous blood depends largely on anatomic conditions. For example, if the right lateral sinus is conspicuously larger than the left, its obliteration will have a more noticeable effect on the outflow of blood than will obliteration of the smaller sinus. This fact is important, but it is not sufficient to explain the papilledema in sinus thrombosis, because in cases of extensive sinus thrombosis papilledema is often absent, but may occur when only the sigmoid sinus is thrombosed. Moreover, the difference in width of the lateral sinuses is more common than papilledema in sinus thrombosis. For this reason, the impediment to the outflow of venous blood per se cannot explain the papilledema in sinus thrombosis unless there is, in addition, inflammation of the dura causing active hyperemia of the leptomeninges and serous exudation into the cerebrospinal fluid. It is likely that venous stasis and serous meningitis also co-operate in causing papilledema after ligation of the jugular vein. As for venous stasis, ligation of the jugular vein may cause an increase in intracranial pressure analogous to that in Queckenstedt's test. This occurs regardless of whether the lateral sinus is obliterated or not. It all depends on whether the inferior petrosal and carotid sinuses are patent or not. Usually they are patent in thrombophlebitis of the lateral

sinus. For this reason, the increased venous pressure caused by ligation of the jugular vein can be transmitted to the cavernous sinus even though the sigmoid sinus is obliterated. This probably explains the frequent failures of the Tobey-Ayer test and the frequent association of abducens paresis and papilledema in these instances.

3. A local compressing force may obliterate or impede the

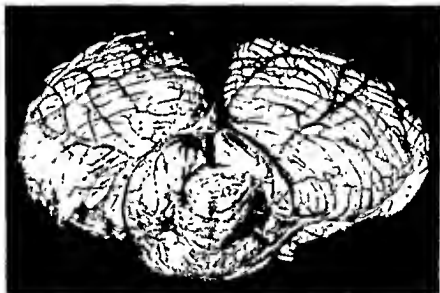


FIG. 18.—Cerebellar cone in a case of brain edema following tonsil infection. There was no brain abscess.

circulation in the veins and capillaries in the immediate vicinity. Moreover, since with expanding intracranial lesions the rise in intracranial pressure is not uniform, even in a single dural compartment, a distortion of the local circulation is added to the effect of compression. The local disturbance of blood circulation may cause brain edema which, in turn, generalizes the increased brain pressure. With brain abscesses, the brain edema is due to a disturbance of the blood circulation as well as to inflammation. The inflammatory component is of greater importance, since extension of the brain edema is not dependent on the size of the abscess. Frequently a small abscess causes edema of the entire

cerebral hemisphere on the involved side, whereas with a large abscess the edema may be restricted to the immediate vicinity of the abscess. Extension of the edema depends on the virulence of the infection, on the *Oedem-Bereitschaft* of the brain, which is more marked in children than in adults, and on the presence of a capsule of the brain abscess.

Both the increase in cerebral bulk and the brain edema may cause dislocations of the brain, called foraminal herniations. The best known foraminal herniation is cerebellar herniation into the foramen magnum (Fig. 18). With this type the edematous cerebellar tonsil and medulla oblongata block the cisterna magna to such a degree that the bony margins of the foramen press on the cerebellar tonsil. This causes the cerebellar pressure cone which may be noted at autopsy in cases of cerebellar abscess and, occasionally, temporal lobe abscess. In cases of cerebellar abscess the roof of the cerebellum is often bulged toward the tentorium, causing herniation of the cerebellar roof up through the tentorial hiatus (Fig. 19). I have never seen a case of this type, probably for want of proper attention. In these cases the edematous cerebellum undoubtedly presses on the cisterna ambiens encircling the midbrain and extending up to the quadrigeminal bodies. The pressure on the cisterna ambiens interferes with the circulation of cerebrospinal fluid and is one cause of the development of internal hydrocephalus in cerebellar abscess.

Herniation into the hiatus, or incisura, tentorii is less well known than that into the foramen magnum. The cerebellar tentorium (Fig. 19) extends from the transverse sulcus of the occipital squama forward to the superior angle of the petrous bones and to the anterior clinoid processes. Between the apexes of the petrous bones the tentorium forms a large foramen, the anterior boundary of which is formed by the dorsum sellae. This foramen is the incisura, or hiatus, tentorii. It is closed by the midbrain, cerebral peduncles and cisterna ambiens. The tentorium covers the cerebellum and forms the base of the temporal and occipital lobes. With tumor or abscess of the temporal lobe the uncus of the temporal lobe may be crowded into the incisura tentorii,

causing a temporal pressure cone which may be seen at autopsy. In such cases, compression of the sylvian aqueduct and obliteration of the cisterna ambiens occur, as with cerebellar abscess. This results in an increase in pressure in the ventricles and thus in dilatation of the ventricles. With temporal lobe abscess the

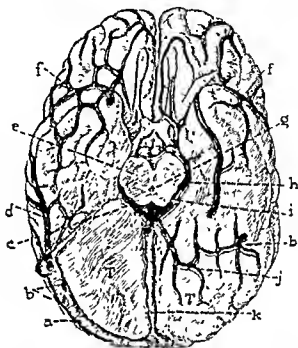


FIG. 19—Inferior surface of cerebrum. *a*, lateral sinus, *b*, lateral occipital vein, *c*, hiatus of cerebellar tentorium; *d*, small anastomatic vein of Labbé, *e*, cerebral peduncle, *f*, middle cerebral vein, *g*, aqueduct of Sylvius, *h*, vein basalis (rosenthal), *i*, cisterna ambiens in the tentorial hiatus, *j*, internal occipital vein; *k*, straight sinus, *T*, tentorium of cerebellum. (Adapted from Bailey.)

ventricle on the involved side is usually compressed; therefore bilateral hydrocephalus cannot occur. However, there is usually hydrocephalus contralateral to the temporal lobe abscess (p. 361). Frontal lobe abscesses of rhinogenous origin rarely interfere with cerebrospinal fluid circulation.

Generalized intracranial hypertension does not cause definite symptoms as long as there is sufficient oxygenation of the brain stem. However, with a disturbance of the blood circulation or

intermittent anemia of the hindbrain, the principal symptoms of increased brain pressure may appear: headache, nausea, vomiting and papilledema. If intracranial pressure reaches a level equal to, or slightly in excess of, diastolic blood pressure and if this rise occurs comparatively rapidly, alterations in pulse rate and respiration and eventually in blood pressure may ensue. The first symptom is the "pressure pulse," caused by irritation of the vagus center in the hindbrain. This is characterized not only by the low rate but by its regularity and the striking tension of the arterial walls. As for the low rate, one must remember that both fever and accelerated and deep respiration have a stimulating effect on cardiac activity, so that under these circumstances a pulse rate of 60-70 may be low. On the other hand, a low pulse rate may exist without intracranial hypertension (p. 356). If there is further increase in intracranial pressure the pulse becomes weak and irregular. This indicates impending paralysis of the vagus center, which may cause a permanent standstill of the heart if there are simultaneous cessation of respiration and sudden fall in systemic blood pressure.

During operation on brain abscess, or spontaneously, symptoms may appear that indicate paralysis of the respiratory center which is not necessarily associated with the finding of a pressure pulse. Respirations become slow and deep, and irregular pauses in respiration occur. This is followed by a rhythmic change of respiratory pause and rapid deep respiration—the Cheyne-Stokes type of respiration. Ultimately respiration ceases entirely.

If there is impending paralysis of the vagus center and the center of respiration, the blood pressure reflex of Cushing (p. 52) may reactivate the failing blood circulation in the brain for a short time. But if intracranial hypertension is not rapidly relieved, the systemic arterial pressure will gradually fall and death will ensue. With high degrees of intracranial pressure the first center which fails is that of respiration, whereas the action of the heart may continue for several hours if artificial respiration is given. In one of my cases of cerebellar abscess the heart beat was noted more than 10 hours after cessation of respiration.

The symptoms of intracranial hypertension do not become manifest in every case at the same time and in the same intensity. Certain constitutional factors determine whether pressure symptoms make a rapid or a slow appearance. For example, in an individual with a great amount of cerebrospinal fluid and large venous channels, it will take longer for the various symptoms of intracranial hypertension to become evident than in a person with a small amount of fluid and narrow venous channels. In the former, more space becomes available in the skull as the venous channels are compressed and cerebrospinal fluid is absorbed. Even more important is the relation between volume of the brain and capacity of the skull. As previously mentioned, the relative size of the brain in infants and children is greater than in adults. However, there are variations in the relation between volume of the brain and capacity of the skull in adults. In old age the brain volume decreases, thus increasing the difference between brain volume and capacity of the skull. For this reason, in children and some adults the symptoms of intracranial hypertension appear more rapidly than in the aged.

Chemistry and immunology of cerebrospinal fluid.—Normal fluid is clear and colorless and shows no pellicle or sediment of any kind. Specific gravity varies between 1.006 and 1.009. Increase in specific gravity indicates the presence of abnormal contents or an increase of normal contents. Normal fluid contains approximately 5 cells per cu. mm. However, from a practical standpoint, the limit between normal findings and definite pathology should be set higher, up to 10 cells per cu. mm. If the counting chamber of Fuchs-Rosenthal is used, the number of cells is usually expressed in terms of thirds, as $8/3$ or $15/3$ cells, since the chamber embraces a space of 3 cu. mm. The total number of cells, therefore, must be divided by 3 to find the number of cells in 1 cu. mm. The number of cells is believed to be greater in the fluid of the lumbar spine than in the fluid of the ventricles, but the difference has no practical importance. Under pathologic conditions the number of cells is greatest close to the involved area. A slight increase in lymphocytes is often

caused by an irritation of the meninges, as it occurs in spinal or cisternal puncture and in encephalography. The cells of the cerebrospinal fluid deteriorate rapidly. For this reason, the fluid should be examined within a half-hour after puncture.

The protein content varies between 13 and 24 mg. per 100 cc.; the plasma protein content is 6-8 Gm. per 100 cc. The protein consists primarily of albumin, globulin comprising only one fifth of the total proteins. Cerebrospinal fluid taken after performing the Queckenstedt test is thought to be somewhat diluted and to contain less protein. The tests generally employed for the determination of protein in the cerebrospinal fluid are Nissl's test, the phase I reaction of Nonne-Apelt and the Pandy test.

The sugar (dextrose) content varies between 40 and 65 mg. per 100 cc.; it is believed to be greater in the fluid of the ventricles than in the lumbar fluid. The total nitrogen content varies between 19 and 22 mg., and the chloride content between 700 and 750 mg. per 100 cc.; the chloride content of the plasma varies between 570 and 620 mg. per 100 cc. The pH ranges from 7.4 to 7.6. The lactic acid content of normal cerebrospinal fluid varies between 6 and 27 mg. per 100 cc. Whether or not normal cerebrospinal fluid contains bactericidal substances is not clear. Some writers claim that the fluid is a good culture medium for bacteria, while others state a contrary opinion.

Methods of obtaining cerebrospinal fluid.—In otolaryngologic practice, spinal puncture, cisternal puncture and encephalography are commonly used.

SPINAL PUNCTURE.—This is performed with the patient in the lateral recumbent position, unless inflation of the spinal canal with air is indicated, when the puncture must be performed with the patient in sitting position to permit ascent of the air into the cranial cavity. The spinal column is arched to widen the intervertebral spaces. The skin is prepared with iodine and alcohol, and all aseptic precautions must be taken to minimize the danger of introducing infection into the spinal canal. General or local anesthesia is seldom necessary, although nervous patients occasionally require general anesthesia.

Spinal puncture needles are 7-9 cm. long and 0.6-0.8 mm. in

diameter, with a stilet to fit the needle. The needle must be sterilized. It should have valves for the measurement of spinal fluid pressure, although this is not an absolute requirement. If there are clinical symptoms of meningitis, a needle with a larger diameter should be used so that, in the event frank pus escapes, it does not clog the needle. The diameter should not be more than 1 mm., to avoid leakage of fluid after the puncture.

A good landmark for guidance of the needle may be made by drawing a horizontal line with tincture of iodine through the vertebral column across the crests of the ilium. The line strikes the interspace between the third and fourth lumbar vertebrae. This is the preferred site for spinal puncture, although the space between the second and third lumbar vertebrae can be used without risk of puncturing the spinal cord. In infants up to 2 years, spinal puncture should be performed either between the first and second or between the second and third lumbar vertebrae. The needle, the tip of which is directed slightly caudad, should be introduced in children exactly in the midline and in adults, 5-10 mm. to one side of the midline.

The needle rapidly pierces the skin, subcutaneous fascia, fat, deep fascia, multifidus muscle and vertebral arches. When the needle touches the ligamentum flavum the speed of advance should decrease. When the needle passes the dura a snap is felt. The stilet is then removed and the fluid allowed to flow directly into the manometer or a sterile test tube. Five to 10 cc. of fluid should be withdrawn for diagnostic purpose. If the pressure is markedly increased, the fluid should be withdrawn very slowly and needles of fine caliber used. The quality of the pulse must be carefully checked during the procedure. If lumbar pressure is very low, the outflow of fluid can be encouraged by pressure on the jugular vein or, with the patient recumbent, by bending the head forward. (With the patient sitting up, bending the head forward does not increase lumbar pressure.) When the needle is withdrawn iodine is applied and collodion placed on the opening in the skin. Unless surgery is to be done, the patient is put to bed immediately after the puncture and kept supine for at least 24 hours to relieve headache and prevent leakage of fluid by decreasing the pressure in the lumbar spine.

Death from spinal puncture has been reported, most frequently in cases of cerebellar abscess, less frequently in cases of temporal lobe abscess. Death in these cases is rarely instantaneous, usually occurring several hours after the puncture. Never-

theless others, as well as the writer, in ignorance of the correct diagnosis, have performed a spinal puncture in cases of cerebellar abscess, and in many such cases the puncture was well tolerated. For this reason one may infer that a fatality occurs when, prior to the spinal puncture, a cisternal block due to compression of the medulla in the foramen magnum was developing and was simply completed by the spinal puncture. This hypothesis is, however, of little practical value, since there are no definite indications of an impending cisternal block. For this reason, every case of cerebellar abscess should be considered one of potential cisternal block and spinal puncture should be omitted. To a certain degree, this holds true for every complication associated with intracranial hypertension, as indicated by papilledema. If, for one or another reason, a lumbar puncture must be performed in such cases, it should be done with the patient in the lateral prone position, with a fine caliber needle, and only 5 cc. of fluid should be slowly withdrawn. In the presence of erysipelas, abscess in the lumbar region or osteomyelitis of the lumbar vertebrae, a spinal puncture cannot be performed.

A common argument against lumbar puncture is that it may cause a rupture of an encapsulated brain abscess or spread of a localized meningitis. With regard to the spreading of a localized meningitis, it is instructive to recall the influence of surgical traumatism on a localized labyrinthitis. The perilymphatic space of the semicircular canals has a structure similar to that of the subarachnoid spaces. A localized inflammation of the perilymphatic space of the semicircular canals, called localized labyrinthitis, can be considered analogous to a localized meningitis. Localized labyrinthitis usually requires mastoid surgery. Because it is usually due to a cholesteatoma of the tympanic cavity, the bone of the mastoid antrum is thickened and sclerotic and there is a fistula in one of the osseous semicircular canals. For this reason, the localized labyrinthitis is subject to considerable mechanical trauma at operation. This injury is certainly more hazardous than that acting on a localized meningitis when 5 cc. of cerebrospinal fluid is withdrawn by spinal puncture. Nevertheless, localized

labyrinthitis seldom spreads after a properly performed radical mastoid operation. Therefore it is scarcely believable that the minor mechanical trauma caused by withdrawal of 5 cc. of cerebrospinal fluid will cause spread of a localized meningitis. Practical experience confirms this conclusion.

So far as rupture of an encapsulated brain abscess is concerned, neither practical experience nor theoretical consideration supports this hypothesis. Of 55 cases of brain abscess observed by the writer, not one pointed to the spinal puncture as a cause of rupture of the abscess. This experience is in agreement with theoretical considerations. According to present knowledge, the withdrawal of 10 cc. of cerebrospinal fluid causes a fall of fluid pressure of not more than 80–100 mm. of water. A simple shift from the recumbent to the erect position causes a fall of ventricular pressure of 100 mm. of water at least, provided there is free communication between the subarachnoid spaces of the brain and the spinal cord. Consequently, for the patient with brain abscess, the shift from the recumbent to the erect position is more dangerous than the withdrawal of 5 cc. of cerebrospinal fluid. Nevertheless, the rupture of brain abscess caused by simple movements of the body is rare, and must be even more uncommon subsequent to spinal puncture.

Another untoward effect of spinal puncture is "dry puncture," i.e., the failure to obtain fluid. There may be various causes. With the needle properly inserted in the spinal canal, a spinal nerve may obstruct the lumen of the needle. A slight movement of the needle or a turn of its long axis may dislodge the obstacle. If particles are clogging the needle lumen, the stylet should be reintroduced and the needle withdrawn slightly. If the flow is very slow, the jugular vein may be compressed. Occasionally the application of an ethyl chloride spray to the thigh will encourage the flow.

When meningitis is treated by daily spinal punctures, in a later period of such treatment puncture may fail to yield fluid. This is probably due to the formation of adhesions in the subarachnoid space of the lumbar cord or in the cisterna magna. In the first instance, puncture must be performed in another ver-

tebral space, above the obliterated space. If the cisterna magna is obliterated, causing a cisternal block, puncture may yield fluid which is xanthochromatic, escapes under low pressure and contains much albumin but only a few cells. This finding is called the syndrome of Froin. In these instances, which are rare in otorhinogenous complications, a cisternal or ventricular puncture must be performed.

Another untoward effect is the mixture of blood and fluid. If there is a large amount of blood the puncture must be repeated in another area or on another day. If the amount of blood is trifling, a slight movement of the needle may remove the blood. If the needle breaks beneath the skin, the fragment should be removed immediately. If it is broken in a deeper tissue layer, its removal must be postponed until x-ray studies permit localization of the needle. Occasionally vascular collapse occurs, particularly if the puncture is performed with the patient in the sitting position. In that event it is not necessary to remove the needle. The patient should be allowed to assume the recumbent position and inhale spirits of ammonia, and then the procedure can be continued. Immediately, or one to two days, after the puncture the patient may complain of pain in the head, neck, shoulder or back; of nausea; of slight rigidity of the neck; of dizziness; of tinnitus, or of pain on micturition. These complaints disappear spontaneously if the patient is kept at rest, and treatment is not necessary. Occasionally the administration of bromides or rectal administration of 5-10 Gm. of magnesium sulfate is helpful. In patients with intracranial complications these sequelae of spinal puncture are comparatively slight, particularly in children; actually the patients frequently feel relieved after the puncture.

CISTERNAL PUNCTURE.—The needle is introduced exactly in the mid-line of the back of the neck, just above the spine of the epistropheus (Fig. 20). In the direct method, the glabella and upper margin of the external auditory canal are valuable landmarks for directing the needle. If these two landmarks are brought in line and this line is continued to the neck, the occipito-atlantoid ligament will be found at this level. After perforation of the occipito-atlantoid ligament the needle immediately enters the cisterna magna. In the indirect method,

the skull is palpated from the external occipital protuberance down to the vertebral spine until the palpating finger meets the resistance of the ligamentum nuchae. The needle, introduced exactly in the midline, is directed toward the occipital squama and pushed up to the squama. Then, by lowering the tip of the needle, the frame of the foramen magnum is approached. Here the needle is pushed

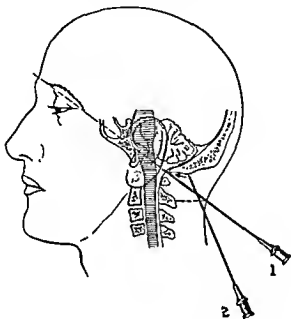


FIG. 20—Cisternal puncture. 1, direct method, 2, indirect method. Arrow indicates direction of the cannula to reach the atlanto-occipital membrane.

through the elastic occipito-atlantoid ligament where frequently, but not always, a snap is felt. I prefer the indirect method.

The puncture may be performed with the patient recumbent or in a sitting position. In the first position, fluid escapes spontaneously, in the second position, it must be aspirated unless an assistant compresses the jugular vein or the patient arrests breathing for several seconds or increases abdominal pressure. After the puncture, the patient should rest for 24 hours, he should not be put flat on his back, but his head should be raised somewhat to avoid positive pressure in the cisterna.

The advantages of cisternal puncture are the lesser degree of headache than after spinal puncture and the possibility of intro-

ducing drugs directly into the cranial cavity. Both advantages are of minor importance in intracranial complications. Moreover, the neck rigidity in meningitis may render the cisternal puncture difficult. For this reason, I perform cisternal puncture only when lumbar puncture is not feasible. The hazards of cisternal puncture are injury to the marginal venous sinus at the frame of the foramen magnum or to another blood vessel and injury to the hindbrain. There is no way of avoiding injury to a blood vessel. The medulla oblongata can be spared if the length of the needle does not exceed 8 cm. ✓

ENCEPHALOGRAPHY.—Both spinal and cisternal puncture gained new indications when encephalography was introduced into otolaryngology. Injection of air into the ventricles of the brain was suggested for differential diagnosis of the various types of hydrocephalus and for the diagnosis of brain tumors. The practical value of encephalography in diagnosis and treatment of inflammatory diseases of the meninges and brain has been demonstrated. ✓

Air can be injected into the ventricles of the brain directly through the skull (ventriculography) or by cisternal puncture (cisternal encephalography) or by spinal puncture (lumbar encephalography). I usually employ lumbar encephalography, occasionally cisternal encephalography, never ventriculography.

For lumbar encephalography, the patient is given 3 gr. of sodium amytal the preceding evening, and the dose is repeated the morning of the examination, without breakfast. Fifteen minutes before the procedure, 1/6 or 1/4 gr. of morphine is given hypodermically. Children are given 1/12 gr. of morphine hypodermically one hour before and 1/2 gr. of sodium amytal one-half hour before the procedure is started. As a precautionary measure against cardiac collapse, some surgeons administer one-half tablet of ephedrine (0.025 Gm.) 30 minutes before introducing the air. General anesthesia is contraindicated because it prevents the entry of air into the ventricles and enhances the risks. Even morphine is rejected by some neurologists who have observed that patients given morphine frequently show a pronounced depression of vital centers which necessitates stimulation during the procedure and are often demoralized so that the injection must be interrupted. The substitution of atropine sulfate for mor-

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phine has been suggested. I have not observed untoward effects of small doses of morphine in intracranial complications.

It is desirable to begin the operation in the x-ray room; when proper facilities are lacking, the patient is taken to the x-ray room after air has been injected. The procedure proper is comparatively simple. Spinal puncture is carried out and cerebrospinal fluid pressure determined with the patient in a horizontal position. He is then directed to sit up slowly, with the head moderately flexed on the trunk. This position is essential to permit the air to enter the ventricles. With the needle in situ, and this is of utmost importance and cannot be stressed too emphatically, the fluid is withdrawn slowly and a lesser quantity of air slowly introduced. I prefer to remove only 5 cc. of fluid at first and then inject 3 cc. of air. Then, again slowly, 10 cc. of fluid is withdrawn and 6-8 cc. of air is injected, and so on. Occasionally the flow of fluid stops before the desired quantity has been removed. In such a case the needle should be shifted slightly or the head bent slightly backward.

Should signs of cardiac collapse appear, the patient must be placed in horizontal position without removing the needle, and stimulants should be administered. Aromatic spirits of ammonia, caffeine sodium benzoate, hypertonic dextrose, adrenalin, pituitrin, digifolin or brandy may be used as indicated. The same measures are carried out when there is complaint of nausea. In the presence of respiratory embarrassment the needle must be withdrawn and inhalation of oxygen started without delay. Once air has been introduced, x-rays must be made at once. The rays should be focused anteroposteriorly, postero-anteriorly and laterally (stereoscopic). It is also desirable to carry out further x-ray studies after 24 hours.

There is no agreement regarding the quantity of air to be injected and the ratio to the amount of fluid withdrawn. The amount of air injected has ranged from 15 to 1,500 cc. I have injected a minimum of 12 cc. and a maximum of 140 cc. of air, the average being 40 cc. Regarding the ratio of air injected to the fluid withdrawn, I usually inject less air, without adhering to any fixed amount, being guided by the tolerance of the individual patient. Usually there has been a difference of 10 cc. between the amount of air introduced and that of fluid withdrawn.

Injection of air into the ventricles is by no means without danger. For ventriculography the mortality rate is about 8.8 per cent, and for lumbar encephalography, about 0.5 per cent. Certain factors render lumbar encephalography rather hazardous. Some patients have such reactions as headache, emesis, chilliness, diaphoresis, restlessness, pallor, backache and circulatory irregularity. To these should be added paresthesias, mental confusion and pains in the thorax and abdomen. These symptoms, or some of them, may last as long as six days, particularly in patients with brain tumor. For these reasons, the procedure should never be carried out to the limit of tolerance. After encephalography one may occasionally note a rise of temperature to 100.4 F., leukocytosis with prevalence of polymorphonuclear cells, an increase of globulins in the cerebrospinal fluid and stasis in the eye grounds. These after-effects usually disappear within four days unless they are caused by the intracranial complication and not by encephalography.

In the presence of intracranial complications one should carefully weigh the indications and contraindications. All clinical and laboratory diagnostic measures should be exhausted before resorting to encephalography, and even then it should be used only when there is no contraindication. In otolaryngology the available diagnostic methods, other than visualization of the ventricles, are so numerous that instances requiring more drastic measures are few and far between. Contraindications to air inflation include: (1) comatose states; (2) pronounced disease of the heart and/or aorta; (3) senility and extreme youth; (4) acute respiratory infections, active tuberculosis and furunculosis; (5) increased intracranial pressure, characterized by papilledema over 2 dipters or lumbar pressure over 300 mm. of water in the sitting position and over 200 mm. in the recumbent position, and (6) suspected complications in the posterior fossa, even in the absence of signs of increased pressure. If, despite contraindications, visualization of the ventricles is absolutely required, the method of choice should be ventriculography.

Frequently lumbar encephalography is indicated in the fol-

lowing conditions: (1) temporal lobe abscess (Fig. 81, p. 362); (2) frontal lobe abscess; (3) complications simulating the clinical syndrome of cerebral abscess, such as the hypertensive phase of serous meningitis (Fig. 64, p. 290), and (4) malignant tumors of the temporal bone and paranasal sinuses.

Despite the repeatedly emphasized dangers of spread of a localized meningitis and rupture of a cerebral abscess, one must consider that injection of air contributes to the maintenance of pressure in the cranial cavity and thereby reduces the danger resulting from decrease in pressure caused by withdrawal of fluid.

I have never withdrawn all of the cerebrospinal fluid, as advocated by some neurosurgeons in cases of brain tumor. It is possible that the x-ray films give better information if the fluid is entirely replaced by air. However, in intracranial complications the only question which encephalography ordinarily is required to answer is whether or not there is an abscess in the frontal or the temporal lobe. Further localization of the abscess is seldom necessary. For this purpose, the technic of encephalography, as described here, is sufficient.

BRAIN

Anatomic relationship between temporal bone and brain.—Both the anterior and the posterior wall of the petrous bone come in contact with the brain. Between bone and brain are the meninges and intermeningeal spaces. If an infection originating in the temporal bone travels into the brain, it necessarily passes through the meninges and intermeningeal spaces. The intermeningeal spaces may be narrow or large enough to form an arachnoid cisterna. Broadly speaking, narrow subarachnoid spaces encourage the extension of an infection into the brain, whereas infections which enter a subarachnoid cisterna may remain there for a long time.

Infections of the tympanic cavity and petrous apex tend to spread into the cerebrum and infections of the internal ear into the cerebellum, whereas infections of the lateral sinus tend to

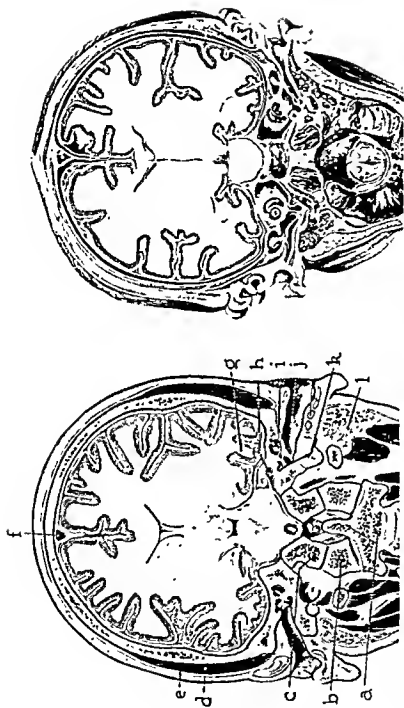


FIG. 21.—Coronal section through brain and temporal bone in adult (*left*) and newborn (*right*) *a*, epistropheus; *b*, atlas, *c*, occipital condyle; *d*, temporal muscle; *e*, fascia temporalis; *f*, superior longitudinal sinus; *g*, temporal lobe; *h*, internal auditory meatus; *i*, tympanic cavity; *j*, external auditory meatus; *k*, cochlea; *l*, parotid gland. Note absence of bony external auditory canal in the newborn. (After Kuerner and Henderich)

spread into the cerebellum and, infrequently, into the cerebrum. For the purpose of exact diagnosis it is necessary to learn which parts of the brain are in topical relationship with the temporal bone. Above the anterior surface of the petrous bone, extending from the anterior surface of the petrous apex to the roof of the mastoid, are the inferior temporal gyrus and the fusiform gyrus of the temporal lobe (Fig. 21). The posterior surface of the petrous bone is adjacent to the cerebellum, and the posterior surface of the petrous apex is close to the pons and the brachium pontis (Fig. 22). The intermeningeal spaces are narrow between the cerebellum and the posterior surface of the petrous bone, whereas between the petrous apex and the pons there are large pontile cisternae (p. 38). Consequently, cerebellar abscesses originating from the posterior part of the petrous bone are not uncommon, but abscesses of the pons originating from the petrous apex are extremely rare. The intermeningeal spaces between the anterior surface of the petrous bone and the temporal bone are narrow (Fig. 21).

Anatomic relationship between nasal and paranasal cavities and brain.—The nasal cavity and paranasal sinuses (except the maxillary sinus) are in topical relationship with the brain. The roof of the nasal cavity is the cribriform plate (Figs. 17, 58 and 59), which is one of the most variable parts of the base of the skull. The width and shape of the cribriform plate depend on the development of the crista galli and the ethmoid sinuses, on the height of the vomer and on the width of the gyrus rectus of the frontal lobe. If the crista galli is thin, the cribriform plate is wide, and vice versa. The plate may have the same width on both sides, but often is asymmetrical. Occasionally the crista galli is bent over one part of the cribriform plate and covers it. The length of the cribriform plate varies between 1.6 and 2.7 cm. and depends on the length of the olfactory bulb. Its thickness varies from fractions of 1 mm. to 2 mm. and is not dependent on the thickness of the rest of the skull. Defects of the cribriform plate are not caused by intracranial hypertension because it is covered and protected by the olfactory bulb. However, the plate

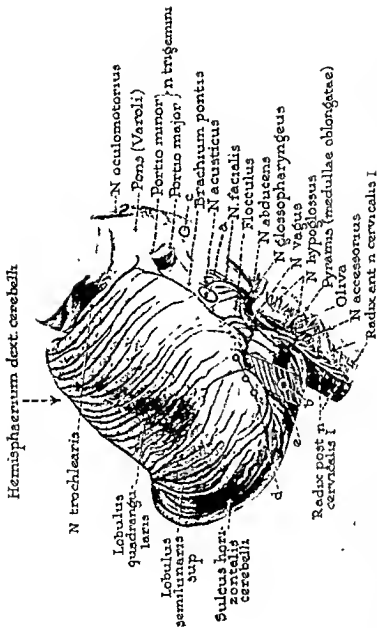


FIG. 22.—Projection of posterior surface of the petrous bone on the cerebellum and brain stem. *a*, internal auditory meatus, *b*, vestibular aqueduct, *c*, apex of petrous bone, *d*, superior angle of petrous bone, *e*, sigmoid sinus (usual location and average width). (Adapted from Alexander.)

not infrequently presents congenital dehiscences, particularly in its anterior part. It is covered by the olfactory bulb and the gyrus rectus of the frontal lobe.

In the area of the cribriform plate the arrangement of the meninges is somewhat complicated (Fig 17). On the posterior

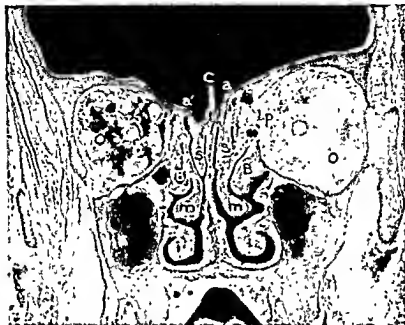


FIG. 23—Coronal section through the orbit (O), lamina papyracea (Lp), bulla ethmoidalis (B), inferior turbinate (i), middle turbinate (m), superior turbinate (s), crista galli (C) and roofs of the ethmoids (a and a'). Note difference in height of the ethmoid roofs on the two sides.

boundary of the plate the dura forms a crescentic fold, the concave margin of which points forward. On the cribriform plate proper, the dura cannot be lifted from the bone but is firmly adherent to the bone and serves simply as periosteum. On the anterior boundary the dura forms another crescentic fold, the concave margin of which points backward. This fold, the tentorium of the olfactory bulb (Trolard), is continued by the dura at the convexity of the frontal pole. The pia-arachnoid covers the olfactory bulb and olfactory filaments (Fig. 17). The filaments with the arachnoid sheaths run into the olfactory mem-

brane, which usually occupies the greater part of the superior turbinate and the opposite part of the nasal septum. With this anatomic structure, an inflammation extending through the cribriform plate into the skull necessarily invades the subarachnoid space. An external pachymeningitis does not occur in this area because the dura is firmly adherent to the bone.

The lateral continuation of the cribriform plate is the roof of the ethmoid which may be, particularly in the posterior portion, in the same level as the cribriform plate. In other instances the roof of the ethmoid rises up to 0.8 cm. above the level of the cribriform plate, particularly in the anterior portion of the ethmoid. The ethmoid roofs on both sides frequently are not on the same level (Fig. 23). This anatomic variation can be visualized on x-ray films in the posterior-anterior exposure. Before endonasal ethmoid operations such films always should be taken to avoid perforation of the roof of the ethmoid (p. 252). The width of the ethmoid roof varies from 0.4 to 1.5 cm. The posterior and middle portions of the roof of the ethmoid are comparatively thick, whereas the anterior portion is thin and occasionally presents congenital dehiscences. Consequently, surgical injuries of the roof occur particularly in the anterior portion of the ethmoid. Occasionally the cribriform plate extends into the ethmoid roof, in which case a few holes can be discovered in the roof lateral to the insertion of the middle turbinate (Fig. 23). Apparently branches of the ethmoid nerves, not olfactory bundles, pass through these holes.

According to the variable relationship of the ethmoid with the frontal and sphenoid sinuses, the length of the ethmoid roof varies considerably. The roof is covered by the basilar portion of the gyrus rectus and, to some extent, by the gyri orbitales (Fig. 24). Between the brain and the ethmoid roof the intermeningeal spaces are narrow. The most posterior cell of the ethmoid may be adjacent to the tuber cinereum of the brain and may extend laterally to the temporal lobe. In rare instances this cell extends to the frontal lobe on the other side.

Owing to the variable size of the frontal sinns, its anatomic

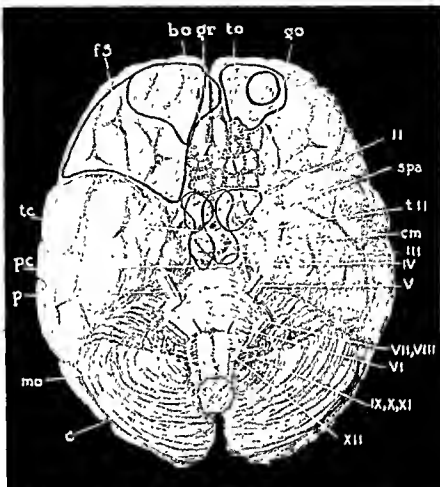


FIG. 21.—Projection of paranasal sinuses on base of the brain. *bo*, olfactory bulb, *c*, cerebellum, *cm*, corpus mammillare, *fs*, Furrow of Sylvius, *gr*, gyrus rectus, *mo*, medulla oblongata, *p*, pons, *pc*, cerebral peduncle, *spa*, substantia perforata anterior, *tc*, tuber cinereum, *to*, tractus olfactorius; *T II*, tractus opticus, *II*, optic nerve, *III*, oculomotor nerve, *IV*, trochlear nerve, *V*, trigeminal nerve, *VI*, abducens nerve, *VII* and *VIII*, facial and acoustic nerves, *IX*, *X* and *XI*, glossopharyngeal, vagus and spinal accessory nerves, *XII*, hypoglossal nerve. Heavy lines indicate projection of frontal sinuses, dotted lines, projection of ethmoid cells, thin lines, projection of sphenoid sinuses (After L. Bergmann.)

relationship with the brain varies considerably. In average cases the posterior wall of the sinus is adjacent to the superior frontal gyrus at the frontal pole (Fig. 25). If the sinus has a marked temporal recess it comes in contact with the superior and middle frontal gyri at the lateral convexity of the frontal lobe. If the sinus has a marked orbital recess it is adjacent to the superior and middle orbital gyri at the base of the frontal lobe (Fig. 24). In rare instances a temporal recess of the frontal sinus extends far back to come in contact with the inferior frontal gyrus. For this reason with a brain abscess originating from the frontal sinus, motor aphasia caused by a lesion of the cortex of the anterior frontal gyrus is not common. If the frontal sinuses present striking asymmetry, the larger sinus may come in contact with both cerebral hemispheres (Fig. 24), and right frontal sinusitis may cause an abscess in the left frontal lobe. The posterior wall of the frontal sinus is always thin, even when the sinus is small. Rarely, a congenital dehiscence is found. The intermeningeal spaces between the brain and the posterior wall of the frontal sinus are narrow.

The roof and lateral wall of the sphenoid sinus come in contact with the meninges and brain. Between the lateral wall and the brain are the cavernous sinuses and optic nerves. Infections which penetrate the lateral wall of the sphenoid sinus must attack these structures before they spread into the brain. Likewise, a large part of the roof of the sphenoid has no direct contact with the brain proper but does have contact with the hypophysis. However, in front of the hypophysis, and occasionally behind it, the roof is in more immediate contact with the brain. In front of the hypophysis is the optic chiasma (Figs. 24 and 25), and in front of the chiasma is the planum sphenoidale, formed by the roof of the sphenoid or, eventually, by the roof of an ethmoid cell overriding the sphenoid. The planum sphenoidale consists of thin bone and is adjacent to the posterior part of the gyrus rectus and tuber cinereum (Fig. 24). Between the planum sphenoidale and the brain is the cisterna chiasmatis (Fig. 26, posterior part of 4). This cisterna becomes involved first if an infection originating in the sphenoid or posterior ethmoid sinus travels through the

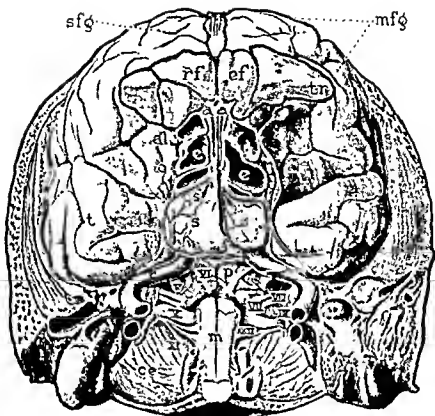


FIG. 25.—Base of brain in relation to paranasal sinuses; base of skull and meninges were removed. *sfq*, superior frontal gyrus, *mfq*, middle frontal gyrus; *rf* and *lf*, right and left frontal sinuses, *aa*, olfactory fissure of nasal cavity; *al*, anterior ethmoid artery; *O*, optic nerve, *e*, ethmoid cells, *z*, ophthalmic artery, trochlear, oculomotor and abducens nerves and first division of trigeminal nerve, *t*, temporal lobe, *oc*, ostium sphenoidale, *s*, sphenoid sinus, *V*, trigeminal nerve; *c*, internal carotid artery, *i*, internal jugular vein, *ce*, cerebellum, *M*, medulla oblongata, *p*, pons, *tr*, temporal recess of frontal sinus, *VI*, abducens nerve, *VII* and *VIII*, facial and acoustic nerves, *IX*, *X* and *XI*, glossopharyngeal, vagus and spinal accessory nerves, *XII*, hypoglossal nerve (After Kilian.)

planum sphenoidale. Exceptionally, the sphenoid sinus extends behind the hypophysis (Fig. 24), and in rare instances it may invade the basilar bone and extend into the posterior cranial fossa. In the latter instance the sinus comes in contact with the anterior pole of the temporal lobe and the basal surface of the brain stem (Fig. 25); but this contact is not immediate because between the brain stem and the sphenoid sinus are the arachnoid cisternae at the base of the skull (Fig. 26). The sphenoid sinuses are often asymmetrical (Figs. 24 and 26), in which case the larger sinus may come in contact with both frontal and temporal lobes.

To sum up, between the frontal and ethmoid sinuses and the brain are narrow meningeal spaces like those between the greater part of the petrous bone and the brain. Between the sphenoid sinus and the brain are arachnoid cisternae like those between the petrous apex and the brain. The anatomic relationship of the cribiform plate with the meninges and brain is similar to that of the internal auditory meatus with the meninges and brain.

Anatomic relationship between pharynx and brain.—Between the nasopharynx and the meninges is the thick bone of the sphenoid body and os basilare. Rarely, there is a patent canal, called canalis craniopharyngeus, which perforates the sphenoid body. Along this canal the embryologic entoderm of the pharynx extends to form the anterior lobe of the hypophysis. The canal is usually obliterated after birth. If it remains patent, the openings are at the highest point of the sella turcica and on the inferior surface of the sphenoid body immediately behind the insertion of the vomer. Here the pharyngeal hypophysis is found. Occasionally the canal becomes very large, harboring a trans-sphenoid cephalocele (p. 34). In such instances there are frequently associated congenital deformities of the skull such as cleft lip and cleft palate. In a few such cases on record, an adenectomy done because of incorrect diagnosis was followed by fulminating meningitis.

The connections of the pharynx with the meningeal spaces and brain by way of the parapharyngeal spaces are especially

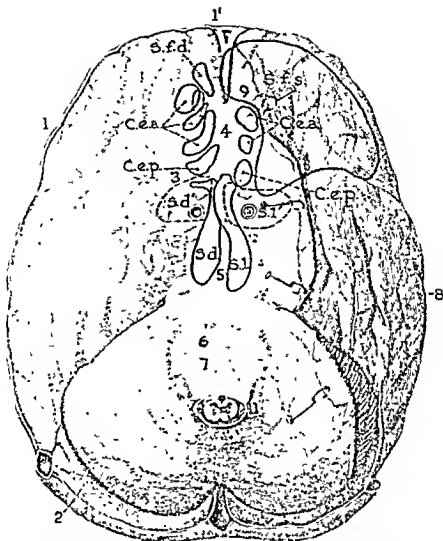


FIG. 26—Projection of paranasal sinuses on the arachnoid at base of the brain. Over the right hemisphere the arachnoid is in place, over the left side it is retracted. *Sfd*, right frontal sinus, poorly developed, *Sfs*, left frontal sinus, extending to right side, *cea*, anterior ethmoid cells, *cep*, posterior ethmoid cells, *sd* and *sl*, sphenoidal sinuses extending in occipital direction, *sd'* and *sl'*, sphenoidal sinuses extending in lateral direction, 1, dura, 1', falx cerebri, 2, tentorium cerebelli, 3, cistern of Sylvius, 4, cisterna corporis callosi, 5, cisterna pontis, 6, arachnoid, 7, arachnoid sheath of cranial nerves, 8, pia, 9, olfactory bulb covered by arachnoid, 10, internal carotid artery, 11, vertebral artery (Adapted from Testut.)

important. On both posterior and lateral walls of the naso- and oropharynx are potential spaces which often harbor infections originating in the pharynx. The different compartments of the retropharyngeal space contain principally loose connective tissue

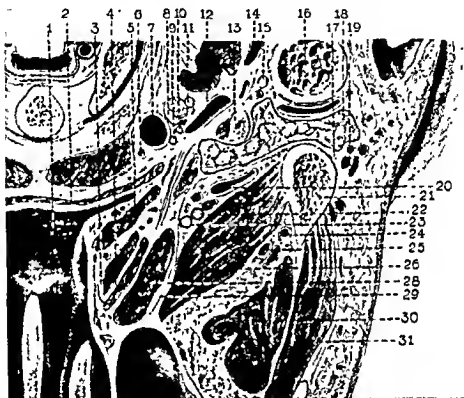


FIG. 27.—Pharyngomaxillary space immediately below base of the skull 1, roof of epipharynx; 2, superior constrictor muscle; 3, pterygoid process, 4, tensor veli palatini; 5, levator veli palatini; 6, ascending pharyngeal artery, 7, internal carotid artery; 8, vagus nerve; 9, glossopharyngeal nerve, 10, spinal accessory nerve; 11, hypoglossal nerve adjacent to superior ganglion of sympathetic nerve, 12, internal jugular vein, 13, styloid process and stylopharyngeal muscle, 14, occipital artery; 15, facial nerve, 16, mastoid process, 17, auriculotemporal nerve, 18, parotid gland, 19, superficial temporal artery, 20, internal maxillary artery, 21, mandible, 22, inferior alveolar nerve; 23, lingual nerve, 24, middle meningeal artery, 25, internal maxillary artery, 26 and 28, external and internal pterygoid muscles; 27, pterygoid process; 30, corpus adiposum (Bichat); 31, masseter muscle. (Adapted from Testut.)

and several lymph nodes. This space does not extend into the interior of the skull and therefore has minor significance in intracranial complications. Of greater importance is the pharyngo-

maxillary space, close to the lateral wall of the naso- and oropharynx. This space consists of an anterior and a posterior compartment. The anterior compartment lies mesial to the mandible. In its inferior portion it is mesial to, and in its upper portion behind, the internal pterygoid muscle (Fig. 27). It extends up to the base of the skull and anastomoses with the dura and cavernous sinus along the pterygoid plexus, the third branch of the trigeminal nerve and the middle meningeal artery (Fig. 27). Inferiorly it ends as a culdesac at the stylomandibular ligament and the upper pole of the submaxillary gland. The middle portion of the anterior compartment contains loose connective tissue, lymph channels, lymph nodes, muscles having their insertion at the styloid process, the internal pterygoid muscles and the parotid gland. The inferior portion contains loose connective tissue, lymph channels, lymph nodes and the glossopharyngeal nerve, and the upper portion (Fig. 27) contains loose connective tissue, lymph channels, lymph nodes, both pterygoid muscles, the muscles of the soft palate, the veins of the pterygoid plexus, the internal maxillary artery with its branches (middle meningeal artery), the third branch of the trigeminal and the parotid gland.

The posterior compartment of the pharyngomaxillary space extends to the base of the skull and anastomoses with the dura, the lateral and cavernous sinuses along the sheaths of the internal carotid artery, the internal jugular vein and the posterior cranial nerves (Fig. 27). Inferiorly it extends to the upper aperture of the chest. Its principal contents are essentially the same at all levels: loose connective tissue, the internal carotid artery, the internal jugular vein, the deep cervical glands and the posterior cranial and sympathetic nerves (Fig. 27).

The following pathways lead from the pharyngomaxillary space to the meninges and eventually into the brain. From the anterior compartment the veins of the pterygoid plexus run into the cavernous sinus, the middle meningeal artery into the dura of the anterior cranial fossa, passing the foramen spinosum, and the third branch of the trigeminal nerve into the arachnoid cisterna of the cavity of Meckel (p. 39), passing the foramen ovale.

From the posterior compartment the internal carotid artery runs into the cavernous sinus, and the internal jugular vein into the sigmoid sinus. From both compartments loose connective tissue extends toward the base of the skull. Infections of this connective tissue (Fig. 54, p. 220) may spread toward the base of the skull to cause osteomyelitis and inflammation of the adjacent dura. Infection originating in the pharyngomaxillary space that reaches the meninges may eventually spread also to the brain, particularly into the poles of the temporal lobes. Cerebellar abscesses caused by an ascending infection along the internal jugular vein and lateral sinus are not known.

Vascularization of cerebrum and cerebellum.—The entire vascular system of the cerebrum extends into the pia and subarachnoid space and spreads over the surface of the brain, including the sulci of the cerebrum. The vascular system of the pia gives origin to the cerebral blood vessels, which form a right angle with the blood vessels of the pia (Fig. 14).

The anterior, middle and posterior cerebral arteries break up into smaller branches until they form an arterial network within the subarachnoid space. Up to this point there seems to be agreement regarding the vascular distribution. The vascularization of the cerebral cortex is, however, controversial. According to the old concept, the arterial network of the pia gives off two types of branches which penetrate the cortex. The first type consists of short cortical arteries which have a very thin wall, break up into smaller precapillaries and capillaries and are nutritive arteries of the cortex. The second type consists of long arteries which penetrate the cortex and, without giving off branches to the cortex, break up into smaller vessels in the subcortical white matter. These are end-arteries; that is, they do not anastomose with each other.

Recent investigations have markedly changed this concept. According to the current view, there are no blood vessels, either arterial or venous, that exclusively serve either the white matter or the cortex. The long arteries, after giving off numerous branches to the cortex, form a dense network in the white mat-

ter. Furthermore, there are short cortical arteries which break up into capillaries within the cortex. There is a free capillary anastomosis in the brain, and, in addition, it is likely that minute vessels of the order of precapillaries anastomose with precapillaries. The blood is carried from the brain by veins which run in two directions, crossing the white matter and carrying blood into the deep veins of the brain stem and the vena galeni, establishing a transverse irrigation of the cerebral hemisphere. Furthermore, there are numerous veins originating in the capillaries of the white matter and running back toward the pia, where they form a venous network. From this network the blood runs into the meningeal veins and finally into the dural sinuses. This arrangement forms a short circuit between the arteries and the veins of the cerebral cortex. In the cerebellum the arrangement of the blood vessels is essentially the same as in the cerebrum.

✓ Electroencephalography.—This procedure consists in the measurement of the electric currents produced by the human brain. Four to six electrodes are fastened to the scalp, usually two or three on each side of the head.

The hair is not cut. The scalp is lightly cleaned over areas 2 cm. in diameter in the frontal, parietal and occipital regions. Into these clean surfaces a bit of ordinary electrocardiographic electrode paste is well rubbed. A 5 or 10 mm. flat solder disk fused to the bare end of a no. 32 enamel wire forms the electrode. The disk is placed firmly on the electrode paste and covered with collodion, which is dried by a compressed air jet or hair drier. The dried collodion holds the electrode firmly to the scalp. The other end of the wire is fastened to a pin jack for insertion into the panel board, connected by a shielded multiwire cable to the recording apparatus. There should be 5,000-20,000 ohm resistance, as measured with a pocket tester, between any two electrodes on the scalp. When the electrodes are in place the pin ends are plugged into the board at the patient's head. The subject is told to close his eyes, relax his facial, jaw and neck muscles and be as quiet as possible.

The recording and amplifying apparatus receive the pulsating electric waves picked up from the scalp electrodes and amplifies them about a million times, so that they are strong enough to move a pen which records the waves on rolled paper. The electric discharges from

the brain appear as wavy ink lines directly on the paper. The speed of the moving recording paper is constant, 3 cm. per second, and usually a timing pen marks off each second. The voltage of the actual brain wave is one twentieth of that of the electrocardiogram, hence the complicated apparatus required to register it.

The normal electroencephalograms of healthy individuals disclose two general characteristics. (1) There are two types of normal brain rhythms, and all persons with a normal central nervous system show varying values for these two groups. The first, or alpha, waves have a frequency of 8-12 per second, with smooth, regular and clearcut patterns and voltages ranging from 30 to 125 microvolts. These waves temporarily disappear when the eyes are opened or if the subject has a problem to work out. The second, or beta, waves have faster frequencies, 19-30 per second, less regular patterns and lower voltages, 10-30 microvolts. They are unaffected by attention or effort. (2) These normal waves are found over all parts of the brain accessible to the surface electrodes on the scalp, the alpha type being more prominent in the occipital region.

In patients with diseases of the brain, particularly when the pathologic process is near the surface, as in the cortex or white matter beneath it, the frequency of the waves, is usually greatly slowed, to 2-5 cycles per second, and the voltage is increased from 75 to 300 microvolts. Less frequently there are abnormal waves of increased voltage in intermediate frequencies, 14-18 cycles, or 28-34 cycles. The abnormal electrical activity in the human brain is distributed into three categories (1) paroxysmal bursts of abnormal waves lasting for seconds to minutes and appearing in a record otherwise normal, seen in epilepsy; (2) generalized, continuous abnormal waves noted all over the brain at all times, seen in trauma, encephalitis and other brain diseases; (3) localized, continuous abnormal waves, found in tumors and abscesses of the brain.

The advantage of electroencephalographic study is obvious: the procedure does not hurt the patient nor does it endanger his life. The disadvantage is that the apparatus is delicate, highly

sensitive and expensive. From the otolaryngologic point of view, it must be emphasized that lesions in the posterior cranial fossa are the hardest to localize by means of electroencephalography. In cases of cerebellar abscess electroencephalography is, as a rule, a failure. The clinical value of electroencephalography in otorhinogenous abscesses of the cerebrum is not definitely established.

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SECTION II

Clinical Aspects

Inflammatory Diseases of the Dura Mater

PACHYMEINGITIS EXTERNA (EXTRADURAL ABSCESS)

PACHYMEINGITIS EXTERNA is an inflammation in and on the external layer of the dura. Since the external layer of the dura serves as the internal periosteum of the skull, pachymeningitis externa is an internal periostitis of the skull.

PATHOLOGY

In all types of intracranial complications a part, or all, of four tissues are exposed to infections: (1) the mucosa of the tympanic cavity or the paranasal sinuses, (2) bone, (3) meninges, and (4) brain. The first tissue to be involved is invariably the bone. The infection may travel through the bone by contiguity or by continuity. In either case the soft tissues of the bone, namely, the blood vessels, periosteum and endosteum, are the first to respond to the infection. The most important are the blood vessels, which are most frequently venous and run from the mucosa to the dura and therefore are called anastomosing blood vessels. A great number of blood vessels of this type run through the tegmen tympani, the floor of the tympanic cavity, the mesial wall of the mastoid antrum, the bony plate of the lateral sinus, the posterior wall of the frontal sinus, the roof of the ethmoid and the roof and the lateral wall of the sphenoid sinus. The infection may travel either within the walls of these blood vessels exclusively or within both the walls and the lumen

of the vessels. In the first instance the condition is called *periphlebitis*, and in the second, *thrombophlebitis*. The infection of the blood vessels, periosteum and endosteum results in *osteitis* or *osteomyelitis*. Osteitis involves bony plates which do not contain marrow, whereas, in *osteomyelitis*, infection of the marrow is the most important feature of the disease.

Infection of the soft tissues may have a striking effect on the

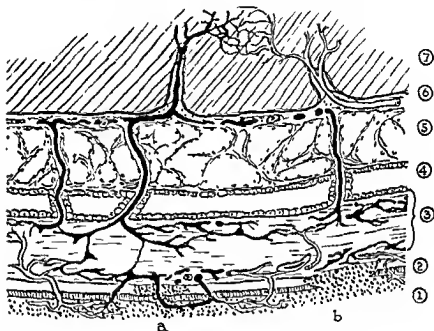


FIG. 28.—Extradural abscesses caused by infection by continuity (a) and by contiguity (b) 1, inflamed mucosa, 2, bone, 3, dura, 4, subdural space, 5, subarachnoid space, 6, pia, 7, brain.

hard tissue of the bone and or marrow, or it may travel through the bone without leaving gross changes. In the first instance the osteitis gives rise to infection of the dura by contiguity, and in the second, to infection by continuity (Fig. 28). In infection by contiguity the inflamed soft tissue causes destructive and constructive changes of the bone. The destructive changes include absorption of bone by osteoclasts or necrosis of bone through thrombosis of the blood vessels. Grossly the bone be-

enues discolored and necrotic and eventually may be entirely absorbed. The constructive changes consist in the formation of osteophytes or sclerosis of bone. Although destructive and constructive changes are usually associated, in the active phase of inflammation the destructive changes predominate and the constructive changes predominate when inflammation is subsiding. When bone involved by osteitis or osteomyelitis is in contact with the dura, the infection may spread to the adjacent dura. This is infection by contiguity and indicates direct extension of the infection through loss of continuity of tissue.

In contradistinction, infection by continuity indicates that the infection travels from the mucosa to the brain along pre-existing channels without causing loss of continuity of tissue, and even without causing gross tissue changes (Fig. 28). Among the pre-existing channels, the anastomosing blood vessels are of utmost importance. Other such channels are: congenital dehiscences of the bone, especially those in the tegmen tympani and cribriform plate; internal auditory meatus; vestibular aqueduct; cochlear aqueduct; facial canal; connective tissue of the subarcuate fossa; sheaths of the olfactory filaments; sheaths of the third branch of the trigeminal nerve; loose connective tissue of the pharyngomaxillary space; veins of the pterygoid plexus; sheath of the great blood vessels of the neck, and, rarely, a patent pharyngocranial canal. Lymphatics play a minor role, except in certain infections which originate in the nasal cavity or pharynx. All these channels are narrow, and when they become involved by infection it is seldom possible to visualize them with the *naked eye*. For this reason tissues harboring infected channels may appear to be grossly normal. To the surgeon, a tegmen tympani or a posterior wall of the frontal sinus may appear to be normal even though the anastomosing blood vessels within these bony plates present perivascular infiltration or thrombophlebitis on microscopic examination.

External pachymeningitis may be caused by inflammation of the (1) tympanic cavity, (2) mastoid process, (3) internal ear, (4) apex of the petrous bone or (5) frontal sinus. It is rarely



FIG. 29A—Section through eustachian tube (*T*) and cochlea (*C*), the latter filled with purulent exudate. *a*, internal layer of dura, separated from external layer (*b*) by artefact, *T_t*, tegmen tympani, *F*, fracture of tegmen tympani, *tt*, tensor tympani, *VII*, geminate ganglion, *c*, marrow space which anastomoses with external dura layer, *ch*, chorda tympani. Inset shows the two dural layers. The internal layer contains numerous veins but no arterial vessels. The external layer contains several small arteries and venules with sparse perivascular infiltration, indicating initial phase of external pachymeningitis due to infection by continuity.

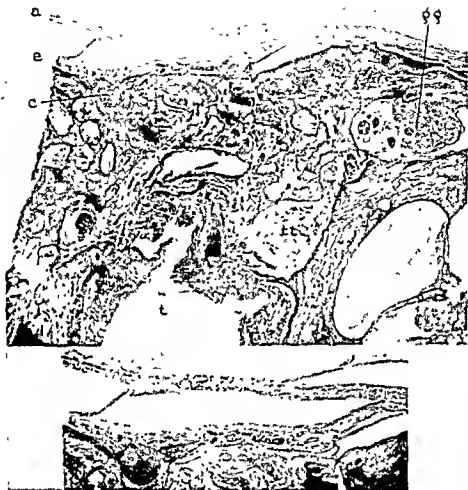


FIG 29B.—Same case as preceding, external pachymeningitis more advanced. Section through eustachian tube (*t*) and cochlea *a*, internal layer of dura, separated from external layer (*c*) by artefact. *T*, tegmen tympani containing numerous pneumatic cells filled with purulent exudate; *F*, fracture of tegmen; *tt*, tensor tympani; *gg*, geniculate ganglion, *c*, marrow space, which contains small artery, thrombosed vein and dense infiltration and anastomoses with external dural layer. (Compare insert.)

due to inflammation of the ethmoid or sphenoid sinus and never to inflammation of the nasal cavity. The infection travels either by contiguity or by continuity (Fig. 28). In the initial phase there is a redness on the outside of the dura caused by inflammatory



FIG. 30.—Section through tympanic cavity (*tc*) and vestibule of inner ear (*V*) filled with pus. *VII*, facial nerve, *tt*, tensor tympani, *st*, stapes, *o*, oval window filled with purulent exudate, *P*, promontory, *rw*, membrane of round window, *a*, osteitis of mesial wall of vestibule, *e*, extradural abscess between dura (*D*) and tegmen tympani (*T*)

hyperemia, and the dura is covered by a layer of fibrin. In advanced cases the dura is covered by granulations which, after a time, may become necrotic. Microscopically the dura presents edema of the connective tissue and a striplike infiltration extending to the endothelium of the dura. The outer layer of the dura is replaced by granulations.

Sometimes frank pus accumulates between dura and bone, forming an extradural abscess (Fig. 30). The formation of such

an abscess depends chiefly on the following factors. (1) Type of infection: in acute otitis caused by pneumococcus type III, extradural abscess is especially common. (2) Attachment of dura to the bone: if the dura is firmly attached to the bone (Fig. 1), a great amount of pus cannot accumulate between bone and dura unless the bone is necrotic. (3) Pathologic changes in bone: if there is a perforation of bone, the purulent exudate produced by external pachymeningitis escapes into the tympanic cavity or frontal sinus. The last-mentioned are the open extradural abscesses that occur with cholesteatoma of the tympanic cavity or osteomyelitis of the frontal bone. In contradistinction are the closed extradural abscesses that occur particularly with infections due to pneumococcus type III. In these cases the necrosis of bone may not exceed the size of a pinhead. Beneath a necrotic area of this size a huge extradural abscess may be hidden. Closed extradural abscesses usually contain more frank pus than open abscesses. Commonly the pus contains the same bacteria as the pus in the tympanic cavity or paranasal sinus.

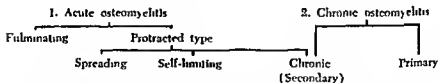
There are different types of otogenous external pachymeningitis, so far as the localization is concerned: (1) lateral pachymeningitis of the middle cranial fossa; (2) mesial or deep pachymeningitis of the middle cranial fossa; (3) lateral pachymeningitis of the posterior cranial fossa (perisinus abscess); (4) mesial or deep pachymeningitis of the posterior cranial fossa.

In general, pachymeningitis of the posterior fossa is more common than pachymeningitis of the middle fossa, and perisinus abscess is more common than deep pachymeningitis of the posterior fossa. Lateral pachymeningitis of the middle fossa (Fig. 30) involves the dura above the tegmen and may extend over large areas of the cerebral hemisphere. It is more often caused by chronic otitis, particularly a cholesteatoma, than by subacute otitis and is almost invariably associated with necrosis of the tegmen tympani and or tegmen mastoideum. In subacute otitis, particularly in children, pachymeningitis is caused occasionally by necrosis of the root of the malar bone. Deep pachymeningitis of the middle fossa involves the dura at the apex of the petrous

bone and is most often caused by subacute otitis media which extends to the apex of the petrous bone, provided the latter is pneumatized. In general, this type of pachymeningitis is not common and does not spread. Perisinus abscess involves the dura mesial to the lateral sinus and is caused by necrosis of the bone in Trautmann's triangle. The necrosis is usually due to acute or subacute otitis. This type does not spread. Deep pachymeningitis of the posterior fossa involves the dura between the internal auditory meatus and the vestibular aqueduct. It is caused by purulent labyrinthitis which has perforated into the posterior fossa or by an infection of the retrolabyrinthine cells, if the temporal bone is well pneumatized. This type is often associated with retention of pus and, for this reason, is the most dangerous type of external pachymeningitis, giving rise to cerebellar abscess and/or meningitis.

Rhinogenous pachymeningitis usually originates in an infection of the frontal sinus. The infection may travel through the posterior wall of the sinus and may cause—usually by contiguity—an external pachymeningitis which commonly remains localized. Or the frontal sinus infection gives rise to osteomyelitis of the skull which, in turn, causes external pachymeningitis. The osteomyelitis which most often causes pachymeningitis is the acute form with protracted course, both the spreading and the self-

CLASSIFICATION OF RHINOGENOUS OSTEOMYELITIS OF THE SKULL



limiting type (see tabulation). Several writers have stated that in the spreading type infection extends more rapidly along the dura than in the bone marrow or external periosteum. In these instances the external pachymeningitis caused by osteomyelitis may extend over large areas of the cerebral hemispheres (Fig. 31). At the base of the skull either the dura is not involved or

there is external pachymeningitis which, however, does not extend beyond the roofs of the orbits. With ear infections, lateral pachymeningitis of the middle fossa also tends to spread over large areas of the hemisphere (p. 95), but the spread is limited to the dura. The bone is not involved, because spreading osteomyelitis



FIG. 31.—Extensive external pachymeningitis (P) following acute spreading osteomyelitis of the skull. *a, a'*, reflected skin flaps; *b*, parietal bone forming a sequestrum; *c*, necrotic bone with numerous fistulas.

of the skull rarely originates in an infection of the tympanic cavity, except occasionally in infants or subsequent to fractures of the temporal bone. In the self-limiting type of osteomyelitis a sequestrum is usually formed, and there is always pachymeningitis beneath the sequestrum.

A girl, aged 9, had osteomyelitis of the frontal bone following scarlet fever. The dura beneath the sequestrum was extremely thin. Beneath the dura was an arachnoid cyst filled with clear cerebrospinal fluid. During removal of the sequestrum the cyst was torn and cerebrospinal fluid escaped. Nevertheless recovery was uneventful.

dural abscess associated with aseptic meningitis is fatal in children. Death is caused not by the infection but apparently by acute intracranial hypertension, indicated by unconsciousness, epileptic attacks and respiratory paralysis. Not infrequently external pachymeningitis is a preliminary phase of a brain abscess (p. 325).

3. External pachymeningitis, particularly in the middle cranial fossa, may undergo spontaneous resolution if it does not extend too far and is not marked. This is proved by the microscopic finding of small osteophytes on the tegmen tympani in temporal bones in which a radical mastoidectomy was performed without exposure of the dura, and by cases in which normal dura has been exposed at a mastoid operation and necessarily becomes infected.

SYMPTOMATOLOGY

Systemic symptoms.—The patient is seldom acutely ill and there is no euphoria. The temperature is normal or only slightly elevated, even with a perisinus abscess. However, the temperature rises immediately when the infection invades the subarachnoid space or a dural sinus. Pulse and blood do not present abnormalities. Even in cases of extensive pachymeningitis and osteomyelitis there is only moderate leukocytosis. The sedimentation rate is normal; in incipient osteomyelitis it may be slightly increased.

General brain symptoms.—Since in pachymeningitis the nerves of the dura are involved, headache is an important symptom. It is continuous, with periodic exacerbations, boring and dull. With perisinus abscess it is often throbbing but, in general, is not as severe as with brain abscess and incipient meningitis.

Headache is an important symptom of all types of intracranial complications but has different significance in acute and chronic infections of the ear and in frontal sinusitis. In acute otitis and acute mastoiditis headache is not outstanding; if it is noted at all, it occurs at the onset of otitis and is mild. Therefore intense headache with acute otitis indicates an intracranial complication, although not necessarily external pachymeningitis, unless

there is nephritis, brain tumor or other disease known to cause headache. In this respect one should not be content with a tentative diagnosis. In ear infections, severe headache must be attributed to the ear unless another cause is proved. This is particularly true for localized headache which, in ear infections, strongly suggests a pachymeningitis. There are different patterns of headache in otogenic pachymeningitis. With perisinus abscess, headache is commonly localized in the occiput or forehead; with lateral abscess of the middle cranial fossa it is localized in the area of the temporal squama or, occasionally, in the vertex. With deep extradural abscess of the posterior fossa headache is localized in the occiput or forehead, and there is often pain in the neck without rigidity. Because of this type of pain or vertigo, these patients often present torticollis. With deep extradural abscess of the middle fossa the gasserian ganglion and branches of the trigeminal nerve may be involved, causing trigeminal neuralgia.

With acute infections of the frontal sinus, headache is prominent regardless of whether the sinus infection is associated with pachymeningitis or not.

Chronic otitis, particularly cholesteatoma of the tympanic cavity, does not cause headache. Therefore headache indicates an intracranial complication or, at least, necrosis of the tegmen tympani. If there is only external pachymeningitis, headache is not severe and may disappear if the pachymeningitis continues for a long period. With chronic infections of the paranasal sinuses, headache has no definite character and is of low intensity. Days and weeks may pass by without headache; but the pain always returns in the same part of the skull. Chronic infections of the sinuses do not cause intracranial complications unless there is an acute exacerbation associated with retention of pus or acute osteomyelitis originating from the chronic sinusitis. With acute osteomyelitis there may be pain as well as headache, both symptoms being of moderate intensity. Headache is due to pachymeningitis, and pain is caused by inflammation of the external periosteum and is referred to edematous regions of the skull.

Tenderness of the skull is uncommon except with pachymeningitis from acute osteomyelitis. Occasionally there is slight tenderness of the temporal squama with lateral abscesses of the middle fossa and of the occipital squama with perisinus abscesses. Percussion of the skull frequently yields useful results. Particularly in lateral abscesses of the middle fossa and in adults, the sound on the involved side is duller than that on the other side. Percussion is simply performed with the finger; special instruments are not necessary. Changes of the optic disks are rare. When present they are probably caused by secondary serous leptomenigitis. Cerebrospinal fluid is normal. With secondary involvement of the leptomeninges, the cell and globulin contents are increased. With perisinus abscess with secondary localized leptomenigitis the cerebrospinal fluid is cloudy but does not contain bacteria. Drowsiness, vomiting and slow pulse are not significant in pachymeningitis, although they occur when the leptomeninges become involved.

Focal brain symptoms.—These symptoms are not present in most cases of perisinus abscesses. However, in a baby, aged 1½ year, with lateral pachymeningitis of the left posterior fossa due to acute otitis, I observed spontaneous rolling movements of the body to the right and ataxia of the left arm. The baby recovered after drainage of the extradural infection. With deep extradural abscesses of the posterior fossa, focal brain symptoms such as dizziness, nystagmus toward the involved side or toward both sides and past pointing with the arm of the involved side are common. The differential diagnosis between deep extradural abscess of the posterior fossa and cerebellar abscess is often difficult. With lateral pachymeningitis of the middle cranial fossa, focal brain symptoms may develop if the abscess reaches considerable size. In these cases paresis of the extremities, epileptic attacks and even nominal aphasia have been noted. The focal symptoms of deep pachymeningitis of the middle fossa are discussed on page 115.

Ear symptoms.—Pachymeningitis may be due to subacute or chronic otitis or purulent labyrinthitis. Frequently the pachymen-

ingitis originates in subacute otitis associated with abundant secretion and sagging of the superior and posterior walls of the external canal. Neither symptom signifies pachymeningitis since both may be due to simple mastoiditis without dural involvement. Sagging of the superior wall of the external canal alone is usually caused by an extradural abscess originating in an infection of the root of the malar bone. These cases are not common. A more significant finding is the definite decrease of systemic and general brain symptoms of acute otitis simultaneously with the appearance of an abundant discharge from the ear, indicating that an extradural abscess has perforated the tympanic cavity. Another suggestive change may take place in acute otitis which has definitely decreased over several days: if, suddenly, more pus escapes from the ear with pulsating rhythm, the tentative diagnosis of extradural abscess can be made, except in infants, in whom this may occur with a simple subperiosteal abscess of the mastoid which has drained into the tympanic cavity.

Acute otitis caused by pneumococcus type III (mucosis otitis¹) is a comparatively frequent cause of pachymeningitis. Apparently the infection of the tympanic cavity may undergo spontaneous resolution, with the drum membrane gray and intact and even the hearing normal. Nevertheless, operation may reveal extensive destruction of the mastoid, a mastoid antrum more or less obstructed by granulations, connective tissue or exostoses and a huge extradural abscess in the middle or posterior fossa. Exceptionally, external pachymeningitis develops in the early stage of acute otitis. In a boy, aged 6, I drained a large area of pachymeningitis of the posterior fossa on the fifteenth day of acute otitis. He had a persistent petrosquamous sinus (p. 26).

In chronic otitis the usual finding is cholesteatoma or acute exacerbation of chronic otitis. In addition, there are clinical symp-

¹The diagnosis of mucosis otitis is not always based on bacteriologic examinations. Frequently the clinical findings permit the diagnosis: gradual onset of the illness; indefinite changes of the drum membrane; marked diminution of hearing, which is not improved by air inflation; marked tinnitus; moderate pain. Later, the inflammation of the tympanic cavity may undergo spontaneous cure, while the infection travels along narrow channels in the bone, particularly in the walls of perilabyrinthine pneumatic cells, toward the meninges.

toms of mastoiditis (edema and tenderness at the tip of the mastoid and sagging of the superior and posterior walls of the external canal). It should be recalled that clinical symptoms of mastoiditis in a case of chronic otitis usually indicate an intra-



FIG. 33.—Puffy tumor of Pott, osteomyelitis of the frontal maxillary process, frontal squama and nasal bones following radical operation on the right maxillary sinus and endonasal ethmoid operation on the right

cranial complication such as pachymeningitis or incipient leptomeningitis (p. 301). Chronic labyrinthitis caused by a cholesteatoma of the tympanic cavity occasionally causes pachymeningitis.

Nasal symptoms.—The diagnosis of external pachymeningitis almost coincides with that of protracted acute osteomyelitis, regardless of whether the infection is progressive or self-limiting. It can reasonably be assumed that in all cases of acute osteo-

myelitis with protracted course and of chronic secondary osteomyelitis (p. 96) there is an external pachymeningitis. In chronic primary osteomyelitis the dura is seldom involved. Acute osteomyelitis usually begins during an acute infection of the frontal sinus or an acute exacerbation of chronic frontal sinusitis or after an operation on the sinuses. In the initial phase there are pain, low grade fever and, occasionally, chills and swelling of the upper

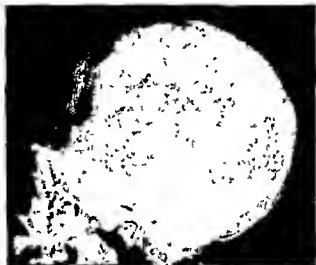


FIG. 31.—Acute spreading osteomyelitis of entire skull vault. Part of the frontal squama was removed. The sequestrum consists of the rest of the frontal squama and part of the parietal bone.

eyelid on the involved side. None is diagnostic of incipient osteomyelitis except when the symptoms appear soon after an operation on the sinuses. If the infection is not arrested the symptoms may subside and the osteomyelitis may pass into a latent phase characterized by evening elevations of temperature, nocturnal pain despite adequate drainage of the sinus and persistent swelling of the upper lid. This phase may continue for a few days, although a period of several weeks is not unusual. Even at this stage the diagnosis of acute osteomyelitis is not definite. The diagnosis becomes evident when the swelling of the skin spreads over the forehead or temporal area beyond the margins of the frontal sinus and causes the puffy tumor of Pott (Fig. 33). There

is no early x-ray evidence of acute osteomyelitis. At this stage the osteomyelitis may run a self-limiting or a spreading course. If self-limiting it terminates in localized pachymeningitis covered by a sequestrum. If the osteomyelitis spreads (Fig. 34), pachymeningitis spreads simultaneously, and the condition terminates in meningitis, brain abscess or chronic secondary osteomyelitis. In the last instance the external pachymeningitis undergoes spontaneous cure, but the osteomyelitis may cause chronic septicemia with multiple metastases in the body.

X-ray examination.—X-ray study does not permit the diagnosis of pachymeningitis, although findings in the bone may allow a tentative diagnosis. In aural cases marked destruction of the tegmen tympani or the bony plate over the lateral sinus indicates eventual involvement of the dura. Such an observation is particularly important in necrosis otitis. In rhinologic cases destruction of the posterior wall of the frontal sinus, a moth-eaten appearance of large areas of the cranial vault (Fig. 34) or sequestra suggest external pachymeningitis.

PROGNOSIS

If otogenous pachymeningitis is properly drained surgically, the outlook is good for lateral abscesses of the middle or posterior fossa, even though the cerebrospinal fluid is cloudy. Systemic diseases such as severe arteriosclerosis and diabetes impair the prognosis. Deep extradural abscesses, particularly those in the posterior fossa, require a guarded prognosis. In rhinogenous pachymeningitis the prognosis depends on the osteomyelitis causing the pachymeningitis. It is favorable in the self-limiting type. In the spreading type it depends on whether it is possible to arrest the osteomyelitis.

TREATMENT

If an extradural abscess is very small and situated above the tegmen tympani spontaneous cure may take place (p. 100). Chemotherapy will hasten cure. Extensive pachymeningitis requires surgery. Likewise, surgery is inevitable when otogenous

pachymeningitis causes perforation of a bone of the skull, because this perforation cannot be considered a spontaneous cure. In lateral abscesses the treatment consists, in acute cases, of a simple mastoid operation, in chronic cases, of a radical mastoid operation, and, in either instance, of exposure of the involved dura.

The mastoid operation is performed with the usual technique. Sharp gonges should be used to avoid concussion of the skull. Exposure of the dura should not be undertaken until the operation on the temporal bone is completed. In lateral abscesses of the middle fossa the inflamed dura should be entirely exposed until normal dura is reached on all sides. Because this is hardly feasible when the pachymeningitis extends far forward toward the roof of the eustachian tube or even toward the greater wing of the sphenoid, the bone should be removed as far as the opening of the eustachian tube unless the pachymeningitis has perforated the greater wing of the sphenoid and the infection has invaded the infratemporal fossa, causing swelling in front of the ear. In this case I do not extend the retroauricular incision to the zygoma, but prefer to make a second incision along the inferior margin of the malar bone to drain the infratemporal fossa.

If the pachymeningitis extends forward but there is no infection of the infratemporal fossa, the tegmen and, eventually, the temporal squama should be removed to the opening of the eustachian tube. This is easily accomplished if a radical mastoid operation has been performed, but it is rather difficult to approach the anterior portion of the tegmen if a simple mastoid operation has been performed. For this reason a radical mastoid operation is often performed, even in cases of acute otitis, to gain access to the anterior boundary of the pachymeningitis. However, in acute otitis a radical mastoid operation should be the last procedure considered because it damages the hearing and leaves the patient with a long-standing aural discharge. In most cases this procedure is not necessary. With good pneumatization of the temporal bone and a high epitympanum, the anterior portion of the tegmen can be approached without removing the

ossicles. If the epitympanum is low, an incomplete radical operation provides sufficient space for removal of the tegmen. In any case, a complete radical operation should be the exception rather than the rule in acute otitis and should be performed only after less radical operations and chemotherapy have failed. After the tegmen and temporal squama are removed to the opening of the eustachian tube, the operation is finished even though it is not possible to reach the anterior boundary of the pachymeningitis. Great care should be taken not to allow a piece of bone to slip into the space between the dura and the base of the skull. The first change of dressing should be made two days after the operation.

If bone is removed beyond the opening of the eustachian tube, branches of the middle meningeal artery may be injured. Hemorrhage from these branches can be arrested by pressing a tampon on the injured vessel for a few minutes. A more annoying injury is that of the superficial temporal artery and auriculo-temporal nerve from a prolonged skin incision. Injury to the artery may be followed by considerable hemorrhage several days after the operation. Because the artery is deeply embedded in the edematous soft tissue it is difficult to arrest the hemorrhage. If the nerve is injured the patient may complain of neuralgias radiating toward the vertex and continuing for several days or even weeks. For this reason the skin incision should not extend beyond the opening of the eustachian tube.

Perisinus abscesses can be immediately recognized when, after removal of the cortex of the mastoid process, pus escapes with pulsatile rhythm. Pulsation is not present if the abscess is adjacent to the inferior knee of the sinus and the pus occupies a large terminal cell at the tip of the mastoid or if the lateral sinus and dura of the posterior fossa are covered by thick granulations. The operation consists of exposure of the involved dura, including the wall of the sinus. Often it is not possible to reach normal dura toward the sacculus endolymphaticus and jugular bulb, but despite incomplete exposure most patients are cured, particularly if young and if chemotherapy is given.

The treatment of deep extradural abscesses of the middle cranial fossa is discussed on page 122.

Deep extradural abscesses of the posterior fossa do not offer a trying problem if caused by purulent labyrinthitis, i.e., if there are complete deafness and nonexcitability of the labyrinth on the involved side. In these cases both the extradural abscess and the labyrinth must be drained by the Jansen-Neumann technic. This operation consists of exposure of the dura of the posterior fossa and the openings of the horizontal and posterior semicircular canals, the latter being used as a guide into the vestibule. If necessary the operation can be extended to the internal auditory meatus.

The surgical problem is more intricate if the abscess is caused by inflammation of the perilabyrinthine pneumatic cells and the inner ear proper is not involved. Here the problem is to drain the extradural abscess without damaging the inner ear. This can be accomplished if the abscess is immediately below the superior petrosal angle. If, however, the abscess is below and/or mesial to the posterior semicircular canal, injury to the inner ear is usually inevitable. The operation is rather hazardous because the opening of a normal inner ear increases the possibility of extension of the infection through the internal auditory meatus into the subarachnoid space. It is likely that chemotherapy may diminish the danger of leptomeningitis in these instances.

In rhinogenous pachymeningitis caused by the self-limiting type of osteomyelitis the sequestrum must be removed. If the sequestrum is in the frontal squama and the frontal sinus is not involved or not developed, the skin and periosteum are incised above the sequestrum and the latter removed. If the sequestrum is in the posterior wall of the sinus and the anterior wall is normal, a Jansen-Ritter operation will permit approach to and removal of the sequestrum. This operation consists of removal of the entire floor of the frontal sinus, associated with broad drainage into the nose. It causes no deformity except the skin scar, because the anterior wall of the sinus remains intact.

When pachymeningitis is due to the progressive type of

osteomyelitis, some surgeons advocate conservative treatment if the condition is not far advanced: incision of scalp abscesses, delay until formation of sequestra and sequestrectomy, even without adequate drainage of the frontal sinus. Personal experience does not justify this conservatism. Of 14 patients with acute osteomyelitis, two treated by this method died of meningitis, in one originating in a thrombosis of the superior longitudinal sinus and in the other in osteomyelitis of the petrous bone. Three other patients were first seen 3, 11 and 13 years, respectively, after onset of the infection. In two, radical operations on the frontal sinus had never been performed; they had been treated solely by sequestrectomy. Both had active foci in the skull and metastases, in one case associated with general amyloidosis. This experience certainly does not favor conservatism. On the other hand, the operation is formidable and associated with severe loss of blood and bad deformity. It consists of complete eradication of the osteomyelitic area and involved frontal sinus, or both sinuses if the infection is bilateral. In this dilemma chemotherapy perhaps offers some hope, although its exact value is not known. However, it is likely that chemotherapy and drainage of the frontal sinus may decrease the frequency of radical operations, provided chemotherapy is used in the early stage of the infection, i.e., when there are puffy tumors but no x-ray evidence of a moth-eaten bone.

PACHYMENINGITIS IN PETROSITIS (GRADENICO'S SYNDROME)

Petrositis is an inflammatory disease of the pneumatic cells or, occasionally, of the marrow spaces in the petrous portion of the temporal bone which surround the internal ear and may or may not extend to the apex of the petrous bone.

ANATOMY AND PATHOLOGY

In well pneumatized temporal bones the pneumatization originating from the mucosa of the epitympanum, hypotympanum and antrum always extends into the petrous bone and produces pneumatic cells which surround the internal ear. These cells

are pericochlear or pericanalicular. The pericochlear cells are located in front of, below and above the cochlea and drain into the epi- and hypotympanum. Those below the cochlea are most frequently present. The pericanalicular cells are located below, above and behind the semicircular canals and drain into the antrum above the horizontal semicircular canal, into the petrosal angle and into Trautmann's triangle. Those below the semicircular canals are most frequently present; they usually anastomose with the cells below the cochlea. All of these cells drain into the hypotympanum in front of, and into Trautmann's triangle behind, the descending portion of the facial nerve. The cells do not form distinct groups; rather they occupy areas which differ considerably in extension and may or may not extend to the apex of the petrous bone. Therefore, if the petrous apex is pneumatized, the perilabyrinthine area and mastoid process are usually pneumatized; but if the petrous apex is not pneumatized, the perilabyrinthine area and mastoid process may be pneumatized. The finding of one large cell in the apex, particularly in the roof of the internal auditory meatus (Fig. 35), when the perilabyrinthine area is not pneumatized is not usual.

The pericochlear cells extend to the apex more frequently than the pericanalicular cells because the distance between the promontory (the bony cover of the basilar portion of the cochlea) and the petrous apex is 8-10 mm., whereas the distance between the antrum and the petrous apex is 15-20 mm. If the cells above the inner ear extend to the apex, they occupy the area above and behind the carotid canal; if the cells below the inner ear extend to the apex, they occupy the area below and behind the carotid canal, and if the cells in front of the cochlea extend to the apex, they occupy the area below and in front of the carotid canal.

The petrous apex is pneumatized in about 30 per cent of normal temporal bones and is diploic in 70 per cent. It presents the same structure on both sides in about 75 per cent of cases and different structure in 25 per cent.

Inflammation of the petrous apex occurs especially in well

pneumatized petrous bones and occasionally in poorly pneumatized temporal bones. In the former the mucosa of the pneumatic cells becomes swollen and the cells fill with frank pus. In progressive cases the bony septums between the cells are absorbed and an abscess containing remnants of mucosa in the pus is formed. The abscess may be open or closed; i.e., it may or may not drain into the tympanic cavity through a fistula. Open abscesses are more common. Multiple abscesses, or one large abscess surrounding the entire inner ear, are rare. In poorly pneumatized temporal bones petrositis is due to osteomyelitis of the petrous bone with abscesses in the marrow. Several otologists believe that even in these cases pneumatic cells were scattered primarily in the petrous bone, which became inflamed. The inflammation, after destroying the walls of the pneumatic cells, invaded the marrow near the pneumatic cells, causing osteomyelitis. This may be true in some instances, but I have noted osteomyelitis of the petrous bone in cases of cholesteatoma of the tympanic cavity and observed it in one case several years after a radical mastoid operation. In instances of this type the petrous bone seldom contains pneumatic cells which could become involved by an infection. For this reason, the osteomyelitis cannot be attributed to an inflammation of pneumatic cells which has spread into the marrow. Furthermore, in a case of tuberculosis of the tympanic cavity, with a petrous apex consisting of marrow spaces and several pneumatic cells, microscopic examination revealed a nonspecific inflammation which was definitely more marked in the marrow spaces than in the pneumatic cells. This proves that in chronic otitis petrositis may be due to primary acute osteomyelitis which may cause rapid infection of the leptomeninges and dural sinuses. In exceptional cases it spreads into the body of the sphenoid or basilar bone or the petrous apex on the other side.

Petrositis is usually caused by acute or subacute otitis. Rarely, an acute exacerbation of chronic otitis causes petrositis, but this is usually osteomyelitis of the apex, which has a poor prognosis. As to acute and subacute otitis, one must recall that in acute

otitis not only the tympanic cavity but all, or almost all, pneumatic cells of the temporal bone are involved, although with different intensity. For this reason, inflammation of the perilabyrinthine, and also of the mastoid cells, should be considered not secondary to acute otitis but an integral part of it. This explains the presence of symptoms of petrositis in the very beginning of acute otitis. If acute otitis undergoes spontaneous cure, in three or four weeks the acute inflammation subsides in both the tympanic cavity and the pneumatic cells of the petrous bone. Under circumstances not clearly understood, the inflammation extends beyond this period and the acute otitis enters the subacute phase. This phase is caused particularly by persistence of infection in the pneumatic cells while the infection in the tympanic cavity is more or less subsiding. For this reason, the purulent exudate produced in the pneumatic cells does not stop draining into the tympanic cavity and through the drum perforation into the external auditory canal. In subacute infections caused by pneumococcus type III, also, the pneumatic cells of the temporal bone, not the tympanic mucosa, produce the purulent exudate. However, frequently in these instances the purulent exudate does not pour into the tympanic cavity because the narrow channels which establish an anastomosis between the pneumatic cells and the tympanic cavity are obliterated by the swollen mucosa or connective tissue. The pressure of pus in the pneumatic cells increases, the bony septums between the pneumatic cells become absorbed and large abscesses form in the pneumatic cells. This type of inflammation, when in the cells of the mastoid process, is called mastoiditis; it is called petrositis when it occurs in the pneumatic cells of the petrous bone. Mastoiditis is more common than petrositis; in fact, petrositis without mastoiditis is rare, but mastoiditis without petrositis is common. This is because the petrous bone is not as frequently well pneumatized as the mastoid and the pneumatic cells of the petrous bone have better drainage toward the tympanic cavity.

According to this concept, it is conceivable that petrositis not only occurs in the very beginning of acute otitis but may be-

come manifest when the acute otitis is believed to be cured, i.e., in the third to sixth week after onset. In these subacute cases the symptoms of petrositis often appear after a simple mastoid operation. If the surgeon fails to establish proper drainage of the perilahyrrhine cells, the infection in these cells may flare up, causing the symptoms of petrositis. Bacteriologic studies show that all micro-organisms which cause acute otitis may cause petrositis, although pneumococcus type III is most common.

In petrositis with acute otitis the pus often pours into the tympanic cavity, causing spontaneous cure of the petrositis. In a few cases the purulent exudate ruptures in one of the following directions.

1. Into the posterior cranial fossa, with formation of a deep extradural abscess (p. 96).
2. Into the middle cranial fossa, with formation of a deep extradural abscess in the area of the petrous apex. Such abscesses are comparatively common because any pneumatic cell above the inner ear may cause them.
3. Into the posterior semicircular canal, endolymphatic sac or internal auditory meatus, with subsequent purulent labyrinthitis. This type of perforation is rare.
4. Into the carotid canal, with subsequent thrombophlebitis of the carotid plexus (p. 24) and cavernous sinus. This type of perforation is rare.
5. Into the jugular foramen, with subsequent thrombophlebitis of the jugular bulb and meningitis. This type of perforation is rare.
6. Into the pharynx, with subsequent formation of a para- or retropharyngeal abscess. This type of perforation, which is comparatively common, may take place through the foramen lacerum anterius or through the jugular foramen. Occasionally the superior cervical glands are involved first, with the lymphadenitis causing an infection of the pharyngomaxillary space. These glands are at the base of the skull, mesial to the internal carotid artery and near the floor of the bony eustachian tube.

In cholesteatoma of the tympanic cavity, petrositis leads rapidly to sinus thrombosis and meningitis. Although there are four sinuses at the petrous apex—superior and inferior petrosal sinuses, carotid plexus and cavernous sinus—usually one or two are involved while the others carry blood.

All of these complications require surgical treatment. Otherwise they cause fatal meningitis. It has been stated that meningitis in subacute otitis is usually due to undiagnosed petrositis. There are only two exceptions: if petrositis due to subacute otitis perforates into the pharynx, spontaneous cure usually ensues even though there is a deep extradural abscess at the petrous apex, and purulent labyrinthitis due to petrositis may undergo a spontaneous cure.

SYMPTOMATOLOGY

Only the symptoms of petrositis proper are discussed, excluding those of eventual meningitis or sinus thrombosis caused by petrositis.

Systemic symptoms.—There may be a slight rise of temperature but no chills or leukocytosis, except in children, who may have fever to 102 F., leukocytosis and even a shift of the blood count to the left. Intense pain and sleeplessness lead to anorexia and fatigue. Petrositis is believed to be more common in males than in females.

General brain symptoms.—These are usually absent or insignificant unless there is a deep extradural abscess, in which case there may be drowsiness, headache and vomiting in addition to the symptoms of petrositis. In one personal case there was thrombosis of a branch of the superior temporal vein of the retina, and in another, hyperemia of the papilla and a concentric defect of the visual field for red and blue.

Focal brain symptoms.—Among the typical symptoms of petrositis are those caused either by irritation or by paresis of the trigeminal nerve. The symptom of irritation, which is more common, is pain. Typical trigeminal pain may immediately follow the pain of acute otitis or develop after cure of the otitis. It con-

continues as long as the petrositis is active, but occasionally disappears while the inflammation is still active. The pain is intense, resembling neuralgia, and occasionally is pulsatile. It either is continuous or appears in attacks, particularly in the night. Pyramidon and salicylates often have no effect and morphine must be administered. Localization of the pain varies. Usually it is localized in the temporoparietal area, less frequently in the face. Sometimes the patient complains of pain in the skull on the involved side. Pain in the teeth and in the depth of, or around, the orbit is characteristic. Not infrequently a tooth is extracted to relieve the pain. When pain is localized in the orbit there are no pathologic changes in the eye other than redness of the conjunctiva and slight epiphora. If the inflamed pneumatic cells extend far into the malar bone beyond the head of the mandible, the auriculotemporal nerve may become involved, with pain in the vertex and occasionally edema above, and in front of, the auricle.

With paresis of the trigeminal nerve the corneal reflex is often absent or definitely diminished on the involved side. This symptom may be present in patients who do not complain of pain of any kind.

The symptoms of irritation of the trigeminal nerve do not permit any conclusion regarding the cause of the irritation. In fact, the pain is the same whether due to a deep extradural abscess, severe irreparable inflammation of the petrous apex or mild inflammation of the apex which may be cured by a simple mastoid operation or even paracentesis of the drum membrane.

Paralysis of the abducens nerve has attracted much interest, although certainly it is not as common as trigeminal involvement. Trigeminal symptoms are commoner because they may be induced by an inflammation either of the perilabyrinthine cells or of the cells of the petrous apex, whereas paralysis of the abducens nerve is usually caused by the rather unusual inflammation of the apex cells proper. If an inflammation of perilabyrinthine cells involves the abducens nerve at all, the cells posterior to the labyrinth are usually responsible. Abducens paralysis may be the only symptom or it may be associated with trigeminal symptoms,

particularly with pain in the temporoparietal area. When both trigeminal and abducens symptoms are associated with acute otitis, the syndrome of Gradenigo is completed. Abducens paralysis alone is occasionally caused by syphilitic meningitis, disseminated sclerosis or a brain tumor. I noted abducens paralysis on the involved side without trigeminal symptoms in two cases 35 days and seven years after a radical mastoid operation. Both patients had an acute infection of the tympanomastoid cavity, and both were cured, although the pathogenesis of the abducens paralysis was not clarified.

More important than isolated abducens paralysis is the association of acute otitis, trigeminal symptoms and abducens paralysis, i.e., the syndrome of Gradenigo. Abducens paralysis usually follows the appearance of the trigeminal symptoms but persists longer, occasionally for months. Abducens paralysis is commonly seen on the involved side, although there are cases on record in which it appeared on the opposite side. In the latter instance the paralysis may be due either to serous or purulent meningitis or to osteomyelitis of the basilar bone and petrous apex on the other side. The syndrome of Gradenigo indicates an inflammation of the cells of the petrous bone extending to the apex. The symptoms do not indicate whether the inflammation is mild and reparable or severe and irreparable, or associated with a deep extradural abscess. To answer these questions, other facts must be considered: the type of otitis, mucosis otitis being more liable to complications than streptococcus otitis; the phase of the otitis when the syndrome develops, i.e., manifestation of the syndrome at the onset or termination of otitis or after a simple mastoid operation; the duration of the syndrome which, calculated from the onset of otitis, is between two and six months. The nervous symptoms of Gradenigo's syndrome may also be caused by leptomeningitis, cavernous thrombosis and thrombosis of the inferior petrosal sinus. However, in these cases there are fever, chills and cerebrospinal fluid changes which point to the basic infection. Only thrombophlebitis of the inferior petrosal sinus may fail to cause symptoms other than abducens paralysis.

Not infrequently, incomplete facial paralysis is noted with petrositis. The paralysis is, as a rule, transitory. In a personal case there was mydriasis on the involved side. Cerebellar symptoms caused by a deep extradural abscess of the posterior fossa are discussed on page 102.

Ear symptoms.—Petrositis may occur (1) during acute otitis; (2) after a simple mastoid operation, (3) during chronic otitis and (4) after a radical mastoid operation.

Petrositis may be caused by all types of acute otitis, and the symptoms may appear either at the onset or at the termination of otitis. Mucous otitis may cause petrositis without causing perforation of the drum membrane. If there is a closed abscess at the apex the otitis may undergo spontaneous cure and hearing may become almost normal, so that only the pain, x-ray evidence and occasionally abducent paralysis point to petrositis. If there is an open abscess at the apex, otitis is said to present the following characteristics: the secretion does not cease for a long time; the discharge is wholly purulent at a time when it should be mucopurulent or mucous if there were not additional petrositis; the discharge is abundant at a time when it should be scanty if there were ordinary acute otitis; sometimes the secretion suddenly ceases for several hours or days and as suddenly returns. Although these symptoms are considered by several authorities to be characteristic of petrositis, I cannot agree because they are to be seen with both petrositis and mastoiditis. These symptoms prove simply that the infection in the tympanic cavity is not localized but has involved other parts of the pneumatic system of the temporal bone. Only when the pneumatic cells of the mastoid process have been eliminated by surgery do these symptoms indicate petrositis, since the mastoid cells can no longer be the source of the discharge.

After a simple mastoid operation, and occasionally several years afterward, petrositis may become manifest. Several otologists have reported that in their experience 90 per cent of cases of petrositis belong to this group. I have not observed such cases as frequently as this. Many cases are due to incorrect indications

for, or a faulty technic in, the simple mastoid operation. Postoperative petrositis occurs if acute mastoiditis was operated on too early, if the surgeon did not thoroughly eradicate the mastoid cells and if a primary suture of the retroauricular incision was done after a simple mastoid operation. In any of these circumstances an infection may occur or pus may be retained in the perilabyrinthine cells, resulting in petrositis. If the petrositis has no outlet, both the mastoid process and the tympanic cavity may undergo uneventful recovery after the mastoid operation. Nevertheless, the petrositis continues. If the petrositis has an outlet, the surgical cavity of the mastoid may be cured but the discharge from the tympanic cavity does not cease.

In chronic otitis, cholesteatoma is of some importance, particularly if there is an acute exacerbation. However, these instances are not as frequent as the cases previously mentioned, and when they occur the symptoms of petrositis are rapidly obscured by those of meningitis or sinus thrombosis.

Petrositis subsequent to radical mastoid operation is exceptional. In some cases, latent osteomyelitis becomes manifest after the operation; in others, osteomyelitis of the petrous bone is due to an acute postoperative infection of the cavity. I saw one case of this type in which spontaneous cure resulted from a perforation into the retropharyngeal space.

Labyrinthine symptoms are not uncommon in petrositis. The symptoms are, as a rule, slight and consist of slight dizziness and spontaneous nystagmus to the other side. Occasionally, diffuse purulent labyrinthitis occurs. In a personal case, an infected pneumatic cell in the roof of the internal auditory meatus ruptured into the left inner ear, causing diffuse purulent labyrinthitis. Although the inner ear was not operated on, the patient was cured (without chemotherapy), but she remained deaf on the left side and the left labyrinth was not excitable (Fig. 35).

X-ray symptoms.—X-ray examination of the petrous bone is of great importance, particularly in closed abscesses of the apex. Although various exposures are necessary to arrive at a definite diagnosis, the Stenvers exposure (Fig. 35) must be considered

the standard. This method discloses the petrous bone of only one side and does not clearly show the perilyabyrinthine cells behind the inner ear since they are covered by the compact bone of the labyrinthine capsule. However, the Stenvers technic presents

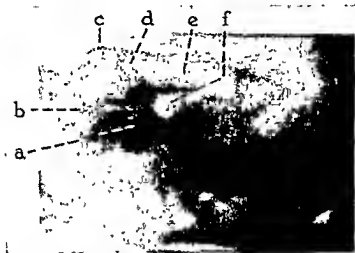


FIG. 35.—Stenvers exposure of left petrous apex. *a*, cochlea, *b*, horizontal semicircular canal, *c*, eminentia arcuata, *d*, superior semicircular canal, *e*, pneumatic cell above internal auditory meatus (*f*) and extending toward petrous apex. The girl, 16, had bilateral otitis media after adenectomy. The right ear was cured spontaneously, on the left, simple mastoidectomy was done. Eighteen days after operation she suddenly had several attacks of vomiting and whirling vertigo. There was third degree nystagmus which changed direction at intervals of several minutes. Caloric test revealed normal excitability of left labyrinth. Two days later she had pulsating pain in left temporal area. Left eyeball was tender. There were diaphanousness, hyperesthesia of skin, Kernig's sign on left, fever of 101.3 F, but no nystagmus, and eyegrounds were normal. Cerebrospinal fluid showed increased pressure, positive Pandy reaction and 150 cells, mostly granulocytes. Dura of the middle cranial fossa was widely exposed and the dura injured. This film was made a month later. Recovery was uneventful, but the patient was deaf on the left and labyrinth unexcitable. No chemotherapy was used.

the structure of the petrous apex exactly and does not disturb the acutely ill patient.

Interpretation of the x-ray film requires much experience. Even the identification of a normal petrous apex may be difficult because the normal apex presents considerable variations of configuration. In one of my cases a deep impressio trigemini was interpreted as a focus of destruction, and an unnecessary opera-

tion was performed. Furthermore, it may be difficult to determine whether a petrous apex consists of small marrow spaces or small pneumatic cells. Moreover, in old persons large translucent spots in the petrous apex often suggest destruction although they are actually due to atrophy of marrow and bone trabeculae. More difficulties may arise in pathologic cases. Cloudiness of pneumatic apical cells is common in acute otitis. Although this indicates an inflammation of the petrous apex it does not allow any conclusions concerning prognosis or treatment. Even when destruction of the petrous bone, characterized by a spot of decreased density with irregular boundaries, and, in addition, a partial defect of the superior angle of the petrous bone are found, the x-ray evidence should always be correlated with the clinical findings. If there is destruction at the petrous apex and if, simultaneously, the clinical symptoms increase, operation on the apex is frequently inevitable. In some cases of mucosis otitis the clinical symptoms recede while the apical infection continues to be active and may cause sudden death from meningitis. In such instances, particularly in the aged, consecutive x-ray studies at various times indicate not only destruction but progressive destruction of the apex. In a case of this type surgery should not be postponed for a long period, regardless of whether the clinical symptoms increase or decrease.

PROGNOSIS

The prognosis for petrositis depends on several factors. Petrositis which develops at the onset of acute otitis usually has a favorable prognosis. The exudate in the apex drains into the tympanic cavity or pharynx, with spontaneous cure. A guarded prognosis must be given when petrositis appears between the third and the sixth week of acute otitis. In such instances the prognosis depends on the following facts. (1) Open petrositis is more favorable than closed petrositis. (2) Accumulation of pus in the roof of the internal auditory meatus implies a greater hazard than accumulation of pus surrounding the eustachian tube. (3) Large accumulations of pus are more favorable than

small accumulations. (4) Petrositis with the complete syndrome of Gradenigo is believed to be more favorable than petrositis without the syndrome. The last statement is correct when petrositis develops at the onset of acute otitis because the diagnosis can be made early. Petrositis which follows a simple mastoid operation is similar to that which occurs during acute otitis. Petrositis due to a cholesteatoma of the tympanic cavity has a poor prognosis. The prognosis for petrositis following a radical mastoid operation is more serious than that for petrositis due to acute otitis but not as serious as that for petrositis due to a cholesteatoma.

TREATMENT

Petrositis is seldom an emergency. It is true that meningitis may develop suddenly. However, prior to meningitis there is, as a rule, a period in which a number of alarming symptoms appear. At this time minor surgical procedures should be tried. This *modus procedendi* is thoroughly justified because petrositis is as dangerous as well as a protean disease and seldom is cured by a single operation.

At first, decompression of the petrous cells should be tried to encourage spontaneous drainage from the pneumatic cells in the petrous bone into the tympanic cavity. In most cases petrositis is cured, the defect at the apex being refilled by calcified connective tissue and newly formed bone. The following procedures may be employed: paracentesis of the drum membrane, simple mastoid,² incomplete radical mastoid and complete radical mastoid operation. It goes without saying that all of these procedures should be supported by appropriate chemotherapy.

The incomplete radical mastoid operation, which can be performed by different techniques, consists essentially of a large opening of the mastoid antrum, partial removal of the posterior wall of the bony external canal, removal of the lateral wall of the epitympanum and plastic repair from the posterior wall of the

²For many years I have performed in these cases, as in cases of subacute otitis, an enlarged simple mastoid operation consisting of removal of part of the lateral wall of the antrum.

membranous external canal. The ossicles and drum membrane remain intact.

Several otologists have abandoned the incomplete radical operation because presumably it does not permit proper search for inflamed pneumatic cells in the petrous bone. Other otologists, including myself, have had good results with this operation, particularly when cells above the cochlea were involved. The fact should be emphasized that a complete radical mastoid operation is often performed because the previous simple mastoid operation failed to relieve pain. In many cases it is performed unnecessarily. An incomplete radical operation would achieve the same result, would save the patient's hearing and would probably arrest the discharge.

The complete radical mastoid operation is essential in all cases of petrositis due to chronic otitis. In cases of petrositis due to acute otitis the complete radical operation frequently must be performed when (1) there are closed abscesses of the petrous bone, (2) the entire tympanic cavity is filled with granulations and (3) there is expectation of inflammation of the pneumatic cells in front of and/or below the cochlea. In the first group, x-ray study indicates destruction of the apex and symptoms are increasing, but there is no discharge from the tympanic cavity. This type of inflammation is usually due to pneumococcus type III. Surgery must advance to the point where the focus of infection in the petrous bone can be properly drained, and for this purpose a complete radical operation is frequently required. In the second group one can hardly expect hearing to be normal regardless of whether a simple or a radical operation is performed. In the third group belong all cases in which both a simple and an incomplete radical operation failed to expose the focus of infection localized in front of and/or below the cochlea. A focus above the cochlea can be drained by the incomplete radical operation provided the epitympanum is high and spacious. If the focus is in front of and/or below the cochlea and is definitely progressive, even a complete radical mastoid operation often fails to provide a satisfactory approach and must be enlarged by

removal of the mesial portion of the anterior and inferior walls of the external canal.

When the focus of infection in the petrous bone is exposed, gentle enrettage of the focus is performed. Forceful curettage may injure the dura or inner ear and may force the infection into the depth of the base of the skull. When the infection has traveled from one apex through the marrow of the basilar bone to the petrous apex on the other side, the question arises whether this was due to the infection per se or to the surgeon's curet.

An operation on the petrous apex is usually inevitable (1) when, despite decompression and entirely adequate chemotherapy, apical symptoms persist; (2) when roentgenographic studies indicate progressive destruction of the apex; (3) when additional symptoms of impending meningitis are noted, including moderate rise of temperature in adults and high rise in children, leukneytosis, increase of cells in the cerebrospinal fluid, vomiting, photophobia, hyperesthesia and tenderness of the atlanto-occipital membrane. Even in these circumstances chemotherapy may be effective in children, but in adults, and especially in the aged, chemotherapy is effective only in association with surgery. It should be emphasized again that an operation on the apex is not a common procedure.

Among the various surgical methods the technic of Ramadier is apparently the best. This technic includes, first, an enlarged radical mastoid operation; i.e., the entire membranous external canal is separated from the osseous canal and the anterior and inferior walls of the osseous canal are removed. The tensor tympani muscle is removed and the semicanal of the muscle is united with the eustachian tube. At the floor of the opening of the tube the carotid canal is opened and the carotid artery carefully exposed with a sharp curet. The exposed artery does not pulsate because actually not the artery but the periosteum of the carotid canal is exposed. Since the carotid plexus occupies the space between artery and periosteum, pulsations of the carotid artery do not reach the periosteum. Injuries of the exposed periosteum are not as hazardous as one might suppose. Slight injuries cause

bleeding from the veins of the carotid plexus, but a tampon firmly inserted will arrest it. According to the literature, the same procedure has been successful in several instances of injuries of the artery. Without having had personal experience, I believe that

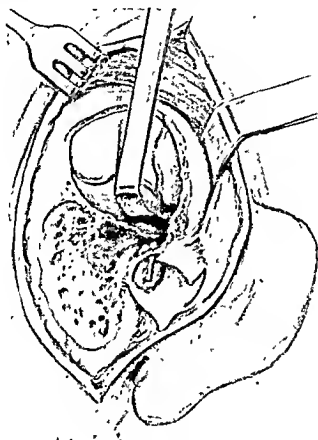


FIG. 36.—Streit's operation

ligation of the common carotid artery would be a safer procedure. When a large opening is made in the carotid canal, a curet is introduced into the apex and the petrositis is drained.

In favorable cases the Ramadier technic permits drainage of the apical abscess and also of a deep extradural abscess provided the abscesses are in free communication. If, however, between the abscesses there is a bony plate, occasionally even the thick cortex of the petrous apex, it would be extremely hazardous to

enter the apical abscess with the curet and perforate the bony plate to drain the extradural abscess. In such cases the apical abscess must be drained by the Ramadier technic and the extradural abscess drained simultaneously by the technic of Streit.

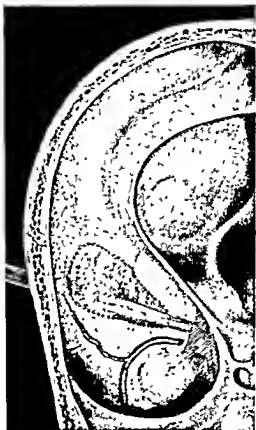


FIG. 37—Subdural approach to the petrous apex, after Ramadier

Another method is to drain only the extradural abscess in the hope that the apical abscess will undergo spontaneous cure. In most cases this is the procedure of choice.

In the technic of Streit (Figs. 36-38) the retroauricular incision is extended forward to the insertion of the helix. The blood vessels and nerves running in front of the auricle should not be severed. Some otologists continue this incision by an auxiliary

incision running from the insertion of the helix perpendicularly upward toward the temporal squama, but my experience indicates that the auxiliary incision can usually be omitted. The oper-



FIG. 38.—Bone defect after Streit's operation.

ation on the mastoid, and eventually that on the tympanic cavity, is then performed according to the clinical indications. The Streit procedure can be performed after either a complete radical or, occasionally, a simple mastoid operation.

When the operation on the ear is completed the temporal aponeurosis is exposed back to the posterior boundary of the temporal squama and the aponeurosis is incised at the inferior

boundary of the temporal muscle. The muscle is pushed aside until the greater part of the temporal squama is exposed with the malar process, the latter forming the posterior and superior walls of the glenoid fossa. To create a proper approach to the apex, most of the temporal squama is removed with a part of the malar process. The capsule of the mandibular joint should not be exposed. The dura and brain are carefully raised and the superior wall of the external auditory canal, the tegmen tympani and the tegmen mastoideum are removed until the eminentia arcuata is reached. By this procedure a triangular defect is created at the base of the skull (Fig. 37). The base of the triangle is in the roof of the external auditory canal and the tip is directed toward the gasserian ganglion. If the operation is properly performed the distance between the tip of the triangle and the gasserian ganglion is 10-12 mm. and the distance between the tip of the triangle and the extreme tip of the petrous bone 20 mm. For this reason, the dura must be raised with the blunt periosteal elevator of Freer for about 10 mm., with the elevator directed not mesialward and forward but straight mesialward; otherwise the dura may be raised at the greater wing of the sphenoid. At this stage the periosteal elevator crosses the anterior surface of the ear canal and the petrosal nerves. If the extradural abscess is found, a drain is inserted into the cavity. I use iodoform gauze for drainage.

The Streit operation has the advantage of providing a short and direct route to the deep extradural abscess. Of greater importance is the avoidance of damage to the ossicles and inner ear. For this reason, any modifications of the technic involving destruction of the entire inner ear, or parts of it, cannot be considered improvements. However, the Streit procedure has several disadvantages. One is that if the dura is raised from the anterior surface of the petrous bone, the geniculate ganglion may be injured, causing facial paralysis, often permanent. To avoid this injury, it is suggested that if the head of the malleus is exposed a line should be drawn, beginning at the head of the malleus and running straight mesialward toward the petrous bone. The

geniculate ganglion lies 1-1.5 cm. mesial to the point where the line meets the petrous bone. In this area the dura should not be lifted unless there is a collection of pus.

Another disadvantage of Streit's operation arises from the firm attachment of the dura to the anterior surface of the petrous bone (Fig. 1) and the occasional presence of pacchionian bodies or herniations of the brain in this area which, when the dura is lifted, may be torn. This results in an opening of the subarachnoid space. Although this is not necessarily followed by infection of the subarachnoid space, injury of the dura should be avoided. It is suggested that the dura be lifted as far as the eminentia arcuata and the periosteal elevator advanced immediately below the superior angle of the petrous bone. In this area the dura is not firmly attached to the bone and there are no pacchionian bodies (Fig. 1). When no pus is discovered in this area the dura is lifted several millimeters beyond the eminentia arcuata. If there is a deep extradural abscess, pus will escape. If no pus is found, either there is no deep extradural abscess or the abscess is near the extreme tip of the petrous bone. In the latter case the abscess will perforate spontaneously after the operation provided the space between the bone and the lifted dura is properly drained with iodoform gauze. If perforation does not follow, an attempt must be made at a second operation to lift the dura toward the apex when the symptoms of impending meningitis continue or are increasing and chemotherapy is ineffective.

A third disadvantage of Streit's operation is that it does not provide sufficient space for eradication of the focus of infection in the petrous apex. This, however, is not serious because the infection often clears spontaneously if the extradural abscess is drained and chemotherapy is applied. The Streit procedure meets with difficulty when the epitympanum is low. However, in such instances pneumatization of the temporal bone is seldom perfect and petrositis rarely occurs. Finally, the Streit procedure does not permit satisfactory drainage of a deep extradural abscess because the brain tends to compress the drain and because drainage is in a horizontal, not a perpendicular, direction. To overcome this

disadvantage, it has been suggested that the pus be aspirated.

INTRADURAL ABSCESS

Intradural abscess is an accumulation of pus within the dura and occasionally between the layers of the dura.

PATHOLOGY

In nearly every case of external pachymeningitis there is infiltration of the connective tissue fibers of the dura. If infiltration

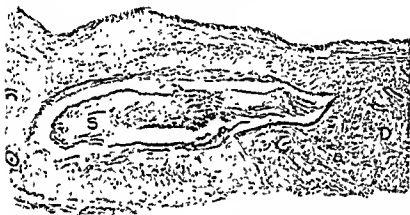


FIG. 39—Saccus empyema due to acute exacerbation of cholesteatoma of tympanic cavity and diffuse purulent labyrinthitis. *D*, dura, *a*, perivascular infiltration, *S*, saccus endolymphaticus filled with pus and fibrin

increases and the leukocytes become disintegrated an intradural abscess may form. Clinically this condition is of minor importance because it is almost invariably associated with leptomeningitis, pachymeningitis externa or interna or brain abscess.

Of major interest is saccus empyema. The endolymphatic sac is embedded in the dura of the posterior cranial fossa and is in free communication with the inner ear, particularly with the sacculus, through the endolymphatic duct. In chronic purulent labyrinthitis there may be an accumulation of pus in the endolymphatic sac, called saccus empyema (Fig. 39). It may also be caused by a purulent infection of pneumatic cells near the posterior surface of the petrous bone or by thrombophlebitis of

the sigmoid sinus, which extends to the endolymphatic sac. In such instances the infection may travel from the endolymphatic sac into the inner ear, causing purulent labyrinthitis. It is likely that this type of infection occurs in patients with a severe intracranial complication and labyrinthitis, although the clinical symptoms of acute labyrinthitis never appear.

The finding of a saccus empyema is not common because it does not persist for a long period. Usually the purulent exudate ruptures through the wall of the sac, causing either a deep extradural abscess of the posterior cranial fossa or a cerebellar abscess.

SYMPTOMATOLOGY

The intradural abscess is not a clinical entity. The symptoms are always obscured by those of meningitis or brain abscess and of labyrinthitis. For this reason, saccus empyema is usually an incidental finding at operation or autopsy.

TREATMENT

The treatment of intradural abscess is that of the basic infection.

PACHYMEINGITIS INTERNA (SUBDURAL EMPYEMA²)

Pachymeningitis interna is an inflammation of the internal layer of the dura which, as a rule, produces a purulent exudate.

PATHOLOGY

Pachymeningitis interna is caused by an infection of the ear, paranasal sinuses or pharynx or by osteomyelitis of the cranium. Commonly the infection travels by contiguity through the tegmen tympani, posterior wall of the frontal sinus, roof of the ethmoid or bones of the skull (Fig. 40), with external pachymeningitis developing first. Infection spreading along dural blood vessels to involve the internal layer causes internal pachymeningitis (Fig.

²C. S. Kubik and R. D. Adams (*Brain* 66:18, 1943) have emphasized that a collection of pus in a preformed space such as the subdural space is not an abscess but an empyema.

32). In these instances surgery discloses gross pathologic changes on the outside of the dura. Less frequently the infection travels along the anastomosing blood vessels from the mucosa of the tympanic cavity or paranasal sinuses to the internal layer of the dura without causing inflammation of the external layer. Surgery

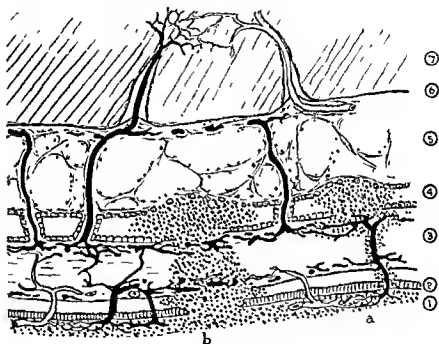


FIG. 40—Subdural empyema due to infection by continuity (a) and by contiguity (b) 1, inflamed mucosa, 2, bone, 3, dura, 4, subdural space, 5, subarachnoid space; 6, pia, 7, brain.

reveals no gross pathologic process on the outside of the dura, but there may be additional infection by contiguity at a site not disclosed at operation.

Another type of infection by continuity originates in thrombophlebitis of the dural sinuses. As was previously mentioned, the dural sinuses receive blood from the dura, especially the internal layer. With thrombophlebitis of a sinus, blood flow in the tributaries is reversed, and the infection is carried from the sinus into the internal dural layer and the subdural space, re-

sulting in internal pachymeningitis. By this mechanism, internal pachymeningitis of the posterior or, eventually, the middle cranial fossa may originate in lateral sinus thrombosis caused by a retrograde infection of the cerebellar and occipital veins; internal pachymeningitis over the parietal and frontal lobes may originate in superior longitudinal sinus thrombosis caused by a retrograde infection of the superior cerebral veins, and internal pachymeningitis of the middle fossa may originate in cavernous sinus thrombosis caused by a retrograde infection of the middle cerebral veins (Fig. 43, p. 155). In none of these instances is there external pachymeningitis and the outside of the dura appears to be normal at operation. In a few cases internal pachymeningitis has been found at a distance from the thrombosed sinus; for example, in the frontal area, while the lateral sinus was involved in the posterior cranial fossa. In these cases the internal pachymeningitis was probably an actual metastasis.

In advanced internal pachymeningitis the internal layer of the dura is changed into granulation tissue covered by pus. Most, or all, of the mesothelial cells of the dura are destroyed. The purulent exudate, which contains various types of streptococci, staphylococci or pneumococci, occupies the dilated subdural space. A serous type of internal pachymeningitis probably does occur but can scarcely be proved because of spontaneous cure. In purulent internal pachymeningitis the arachnoid is nearly always involved either by contiguity or along the paechnionian bodies. The inflammation is mild and localized unless the internal pachymeningitis is virulent and causes spreading leptomeningitis, which usually is fatal. The cerebral cortex beneath the subdural empyema is anemic and the veins of the pia may be dilated. Frequently the cortex is flattened or depressed. Necrosis of the underlying cortex, forming a cortical brain abscess (p. 327), is not uncommon and probably is responsible for the focal brain symptoms.

The external layer of the dura usually presents only slight perivascular infiltration when the internal pachymeningitis was due to infection by continuity. With infection by contiguity both

layers may be changed into granulation tissue so that the dura consists of granulation tissue throughout. In some instances the infection produces less granulation tissue but causes thrombosis of the blood vessels of the dura, resulting in discoloration and

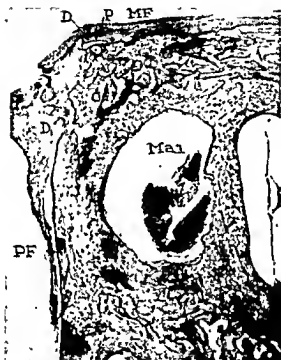


FIG. 41.—Internal pachymeningitis (*P*) due to acute exacerbation of chronic otitis. *Mai*, internal auditory meatus, *MF*, middle cranial fossa; *PF*, posterior cranial fossa, *O*, osteitis of superior angle of petrous bone, *D*, dura; *D_p*, markedly thickened dura; *F*, dural fistula (After Grabscheld)

necrosis of the dura. Since the two vascular layers of the dura anastomose with each other, necrosis will result only when both layers are involved. For this reason, when a necrotic spot is discovered on the outside, one can reasonably assume that the necrosis involves the entire thickness of the dura. Later the necrosis gives way to a fistula communicating between the subdural space and an extradural abscess or, eventually, the cavity of the middle ear or a paranasal sinus.

Internal pachymeningitis is most frequently found at the base

of the temporal lobe (Fig. 41), or on the anterior surface of the cerebellum if it originates in the ear; it is found at the base or on the anterior and lateral surfaces of the frontal lobe if it originates in the paranasal sinuses or pharynx, and it may be found almost anywhere in the brain if it is caused by metastasis.

If internal pachymeningitis produces a serous exudate or only a small amount of pus, spontaneous cure is likely, with adhesions between dura and arachnoid, originating principally in the dura. In more advanced cases a fistula of the dura may form. It must be kept in mind that such a fistula may be caused by internal pachymeningitis, a brain abscess or both (Fig. 41). In neither instance does the fistula establish adequate drainage, and if drainage is not established surgically, leptomeningitis is likely. Occasionally subdural empyema spreads over large areas of the brain. Up to 100 cc. and more of pus has been found covering the hemispheres from the occipital to the frontal pole. Meningitis is the final stage unless there is surgical intervention.

SYMPTOMATOLOGY

Pachymeningitis is not a well defined clinical entity, for the symptoms frequently are obscured by those of sinus thrombosis, brain abscess, labyrinthitis or osteomyelitis. For this reason diagnosis is often difficult, and internal pachymeningitis is probably more common than clinical diagnoses indicate.

The disease may take a fulminating course, lasting a week or less, or a protracted course, extending over several weeks. As in pachymeningitis interna haemorrhagica, the fluctuation and variety of symptoms are significant in pachymeningitis interna purulenta with a protracted course. The patient may appear to be acutely ill, with fever, drowsiness, pareses and cloudy cerebrospinal fluid, then several days later show definite improvement of all symptoms. Unless proper treatment is administered, the improvement might turn rapidly into fatal meningitis.

Emphasis is frequently laid on the latent course of a large subdural empyema. However, analysis of "latent" pachymeningitis interna in retrospect often reveals that it was not latent,

but rather that there was failure to evaluate the symptoms properly.

In a boy, aged 19, who presented what seemed to be a simple cholesteatoma of the left tympanic cavity, a typical radical mastoid operation was performed. The tegmen was necrotic and was removed. The dura of the middle fossa was hyperemic, bulged toward the epitympanum and was pulsating. The night after the operation meningitis developed and took an extraordinarily fulminating course, with death the following day. Autopsy revealed marked pachymeningitis interna on the involved side and leptomeningitis.

In this case the internal pachymeningitis was apparently latent. However, in retrospect it became evident that there were symptoms prior to operation: the patient complained of headache, more marked than with a simple cholesteatoma associated with tegmen necrosis, which gradually increased and was localized in the left occiput. Furthermore, two months before admission he had two "sleeping spells," during which speech was strikingly altered. On admission the patient was acutely ill and percussion over the left temporal squama revealed a dull sound. The tip of the mastoid was definitely edematous. Therefore, internal pachymeningitis was not actually latent, although correct diagnosis was not made. A brain abscess was considered before operation, but lumbar encephalography did not reveal pathologic changes. The cerebrospinal fluid was normal except for a positive Pandy reaction.

Although in some instances internal pachymeningitis presents a meager symptomatology, in others there is a great variety of symptoms. As with brain abscess, the general brain symptoms are of utmost importance although the intensity fluctuates, whereas it is continuous with brain abscess.

Systemic symptoms.—The patient, usually male, is acutely ill. The temperature is only slightly raised, to 100.4 F. or more, unless there is meningitis or sinus thrombosis. The pulse rate is slightly increased, according to the temperature. The blood contains 15,000–30,000 leukocytes.

General brain symptoms.—The principal symptom is headache, which may be localized in the temporal squama on the involved side, in the occiput or in the frontotemporal area. Vomiting, with or without previous nausea, is common. The patient is

drowsy, but several days later may be alert and clear. Later there may be delirium, stupor and coma. Papilledema as well as other symptoms of intracranial hypertension occurs infrequently, and optic neuritis occasionally. Meningeal symptoms, such as rigidity of the neck and Kernig's sign, are not striking unless there is incipient meningitis. However, marked meningeal symptoms are sometimes noted at the onset; they may recede in several days, to be replaced by marked focal symptoms. Several days later the meningeal symptoms may reappear, indicating fatal meningitis.

The cerebrospinal fluid is cloudy and contains a large amount of leukocytes, albumin and globulin but no bacteria. Pressure is increased and the sugar content normal. If the course is protracted, the findings in the cerebrospinal fluid may improve considerably while the clinical symptoms are stationary or even increased. One should not be misled by these contradictory findings, called *syndrome de discordance*.

Focal brain symptoms.—Since internal pachymeningitis may occupy various sites on the surface of the brain and have an intermittent clinical course, the focal brain symptoms are extremely variable and fluctuant. Common symptoms are: pareses of the contralateral extremities (rarely of the ipsilateral extremities); jacksonian attacks; nominal aphasia; motor aphasia; hemianopia; paresis of the abducens nerve; paralysis of conjugate movements of the eyes, usually to the contralateral side, and associated or unassociated with deviation of the eyes toward the involved side; ptosis; difference in size of the pupils, the larger usually being on the involved side, and paresis of the tongue. If due to pressure on the cerebral cortex they may be temporary. If due to a cortical abscess they are permanent.

Aural and nasal symptoms.—Pachymeningitis interna is most often seen in chronic otitis media with cholesteatoma and rarely without cholesteatoma. In acute otitis there is primarily a sinus thrombosis which causes internal pachymeningitis in the posterior or middle or anterior cranial fossa.

An exceptional case was that of a woman, aged 50, who presented acute otitis on the right side of two days' duration. Examination of the

drum membrane revealed recent acute otitis; nevertheless, the patient was acutely ill, with temperature 101.3 F. Paracentesis of the drum membrane caused vascular collapse. Two days later she was hospitalized with generalized meningitis, hemolytic streptococci in the spinal fluid and paresis of the left arm. At operation a small sequestrum was found in the tegmen tympani. Beneath the sequestrum was a fistula of the dura through which pus escaped. There was also periphlebitis of the lateral sinus but no thrombosis. Granted that the history as reported by the intelligent patient was correct, internal pachymeningitis was established with extraordinary rapidity in this case.

Pachymeningitis caused by labyrinthitis is rare. Even more unusual was that in a case in which a retroauricular fistula was closed by a plastic procedure 13 years after a radical mastoid operation. After operation internal pachymeningitis developed, followed by fatal leptomeningitis.

Pachymeningitis interna originating in the paranasal sinuses is most often due to chronic frontal sinusitis causing necrosis of the posterior wall of the sinus. In acute infections of the frontal sinus the micro-organisms may travel along the anastomosing blood vessels toward, and eventually through, the dura. Infrequently, internal pachymeningitis originates in a chronic inflammation of the ethmoid. There are a few reported cases in which it was caused by acute ethmoiditis. In all, there was orbital swelling on the involved side. Obviously all paranasal sinuses, even the maxillary sinus, may cause metastatic pachymeningitis interna if they are the primary source of septicemia. Pachymeningitis interna may also be caused by acute osteomyelitis originating in the frontal sinus. In such cases it is apparently not the external pachymeningitis but the infection of the superior longitudinal sinus which causes the internal pachymeningitis.

In a personal case there were extensive osteomyelitis of the frontal squama, thrombophlebitis of the superior longitudinal sinus and extensive pachymeningitis interna. At autopsy a great amount of pus, containing *Staph. albus*, was found between the occipital lobes.

DIFFERENTIAL DIAGNOSIS

Great difficulties are encountered in the differential diagnosis of pachymeningitis interna and brain abscess. In fact, it is usu-

ally impossible to arrive at a definite diagnosis after a single examination. In some cases the fluctuation of symptoms, the early manifestation of marked focal brain symptoms indicating involvement of the cerebral cortex, e.g., motor aphasia or jacksonian attacks, and the absence of intracranial hypertension and marked leucocytosis favor the diagnosis of subdural empyema. More often the correct diagnosis is made at operation. If pus escapes through the incision in the dura, puncture of the brain should be postponed unless the diagnosis of brain abscess is firmly established. Puncture of the brain should also be postponed when a dural fistula is disclosed because it may be caused either by a brain abscess or by subdural empyema.

Differential diagnosis of external pachymeningitis and external plus internal pachymeningitis is difficult. The following observations may be helpful: patients with external pachymeningitis are not acutely ill; the general and focal brain symptoms are not marked, if present at all; there is no fluctuation in intensity of symptoms; the cerebrospinal fluid is clear, and the dura shows neither necrosis nor fistulas.

Differential diagnosis of subdural empyema and purulent meningitis is extremely difficult, especially when the former runs a rapid course. In the presence of meningeal symptoms the predominance of focal brain symptoms pointing to involvement of the cerebral convexity and the continuous absence of bacteria in the cerebrospinal fluid suggest the diagnosis of subdural empyema.

PROGNOSIS

The outlook for internal pachymeningitis is not favorable. Among 50 cases, the mortality rate was 65 per cent. The outlook is practically hopeless for cases of cholesteatoma plus sinus thrombosis plus internal pachymeningitis. It is extremely hazardous also when internal pachymeningitis runs a "latent" course and even aural examination fails to reveal an acute exacerbation of chronic otitis. In these instances, if a radical mastoid operation or an operation on the paranasal sinuses is performed without creation of sufficient drainage of the subdural empyema, ful-

minating meningitis may follow. In the case mentioned previously (p. 136), the dura of the middle fossa was simply hyperemic when exposed during the radical operation, but a day later autopsy revealed necrosis of the dura of both middle and posterior fossas.

TREATMENT

Except in mild types of internal pachymeningitis, which are rarely diagnosed and probably undergo spontaneous cure after elimination of the bone infection, the treatment is always surgical. Exposure of the dura alone may be sufficient for internal pachymeningitis of the posterior fossa caused by a sinus thrombosis, particularly in young persons. In all other cases the subdural space must be drained. The question arises whether this should be done by incision or excision of the dura. Although it is difficult to keep a simple incision open if no drainage tube or gauze is introduced into the subdural space, excision may sever adhesions between dura and arachnoid which protect the subdural space. For this reason, I prefer the following procedure. If there is necrosis or a fistula of the dura or if the diagnosis of internal pachymeningitis is firmly established, the dura is incised by a cross-like incision. The four dural flaps are reflected and a large opening of the subdural space is created. Adhesions in the subdural space should be left untouched. If there are no adhesions, a probe should not be introduced into the subdural space to locate adhesions. In the absence of adhesions, the length of the cross-like incision corresponds to the findings in the cerebral cortex, which is anemic and edematous and presents dilated veins in the area of subdural empyema. In any event, the incision should not be too small, because the edematous brain often bulges through the opening in the dura and the herniation through a narrow opening may become necrotic. The exposed brain is covered with vaseline gauze, the wound left open and chemotherapy administered. If, later in the course, the diagnosis of subdural empyema proves to be incorrect and brain abscess is diagnosed, the brain can be punctured at the previously exposed site or through a sterile field.

If internal pachymeningitis is widespread and cannot be properly drained through the mastoid or frontal sinus, one or several trephine openings must be made, preferably in the fronto-temporal and occipital areas. Chemotherapy will hasten recovery in mild cases, and in severe cases is a useful adjuvant to surgery.

PACHYMEINGITIS TUBERCULOSA

Tuberculosis may involve either the internal or the external layer, but only exceptionally both layers of the dura simultaneously.

Tuberculous internal pachymeningitis is commonly caused by tuberculous meningitis. Small tuberculous nodules are found on the inside of the dura in the area of the sella turcica, on the clivus and in the area of the lesser sphenoid wings and optic foramen. From a practical point of view, this type of tuberculous pachymeningitis is of minor importance. The more interesting type is tuberculous external pachymeningitis from infection by contiguity originating in tuberculous otitis media, tuberculous osteomyelitis of the malar process or temporal squama or, rarely, tuberculous infection of the ethmoid or sphenoid. In these cases there are diffuse or nodule-like infiltrations, ulcerations and/or granulations on the outside of the dura. Rarely, the tuberculous inflammation extends to the internal layer of the dura; however, in a few cases on record the inflammation traveled through the dura and pia-arachnoid and even extended into the brain.

The diagnosis of tuberculous pachymeningitis is difficult. If it is due to tuberculous leptomeningitis, the diagnosis is impossible and unnecessary. If it originates in tuberculosis of the temporal bone, the constant headache and normal cerebrospinal fluid may point to tuberculous external pachymeningitis.

The prognosis for tuberculous external pachymeningitis is unfavorable if associated with tuberculosis of the lungs or skeleton. In the absence of active tuberculosis in the body, pachymeningitis tuberculosa may continue for several years, even though surgery is not performed on the ear.

Treatment is surgical if there is no more active tuberculosis

in the lungs or skeleton. It consists of a radical mastoid operation and exposure of the dura. In several reported cases cure followed surgery and postoperative x-ray therapy. If there is active tuberculosis in the lungs or skeleton, surgery is not to be considered.

PACHYMEINGITIS HAEMORRHAGICA INTERNA

Internal hemorrhagic pachymeningitis is due to a primary proliferation of the submesothelial tissue, probably after a lesion of the dural mesothelium. The proliferation is associated with exudation of fibrin and hemorrhages. When the blood becomes organized in a later period, membranes are deposited on the inside of the dura.

In several cases of ear diseases internal hemorrhagic pachymeningitis has been noted, but these findings seemed coincidental. The otogenous origin of internal hemorrhagic pachymeningitis is not proved.

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Inflammatory Diseases of the Dural Sinuses; Septicemia

PERIPHLEBITIS AND THROMBOPHLEBITIS OF DURAL SINUSES; SEPTICEMIA

PERIPHLEBITIS is inflammation of the wall of the sinus which originates in the surrounding tissues and involves the external layer of the sinus wall. Thrombophlebitis is inflammation of the wall of the sinus which originates in the surrounding tissues, involves the entire thickness of the sinus wall and causes localized destruction of the endothelium. In the area where the endothelium is destroyed, the sinus is partially or completely occluded by an intravascular clot.

PATHOLOGY

Inflammation in the temporal bone, paranasal sinuses or pharynx may cause thrombophlebitis of the dural sinuses or veins of the neck. Thrombophlebitis of the dural sinuses may, in turn, cause secondary complications such as internal pachymeningitis, leptomeningitis, brain abscess and septicemia. Occasionally thrombi are found in the lateral and/or superior longitudinal sinuses without inflammation of the walls. These clots occur particularly in aged or debilitated individuals with an infection such as pneumonia or erysipelas or with a malignant tumor. They are called marasmic thrombi. This type of thrombus is not common and is not to be considered an intracranial complication and therefore is not discussed in this volume. It should

be emphasized, however, that in children marasmic thrombi may be associated with septic thrombophlebitis. Owing to this association many of the dural sinuses of the skull may be occluded.

There are four ways by which an infection may cause thrombophlebitis of the sinuses and veins of the neck: (1) infection by contiguity; (2) infection by continuity; (3) infection by compression; (4) infection by injury.

1. *Infection by contiguity.*—This is the commonest route, especially when the lateral sinus is concerned. For this reason the following discussion deals particularly with the lateral sinus. The bony plate covering the sinus is absorbed by osteitis and replaced by granulations and pus. Thus the sinus is covered by granulations and purulent exudate, but there are seldom necrotic areas. The granulations may proliferate to such a degree that the sinus becomes compressed and contains only a thin column of blood. These changes indicate a periphlebitis analogous to external pachymeningitis. Since the periphlebitis may continue for a comparatively long period without causing thrombophlebitis, it must be considered both a pathologic and a clinical entity.

FORMATION OF THROMBUS.—When inflammation of the sinus wall involves the endothelium, platelets are deposited on the injured vascular surface and the periphlebitis becomes a thrombophlebitis (Fig. 42). I agree with the many otologists who believe that injury of the endothelium is the principal cause of thrombus formation in the dural sinus. Others claim that a thrombus may be formed by alteration of the blood in the area of the inflammation owing to a specific action of bacterial toxins. This alteration may be of great importance in postoperative and static thromboses as they occur in general surgery, but it is of minor importance in otogenous and rhinogenous thrombophlebitis of the dural sinuses. This opinion is based on the numerous cases of mastoiditis in infants in which granulations and frank pus are found adjacent to the sinus wall but a thrombus seldom forms, even though the dura of infants is more permeable than that of adults.

Additional factors encourage the formation of a thrombus, particularly in the lateral sinus. The blood pressure in the sinus

is low. From the tributary veins, blood pours into the sinus at various rates, thus causing eddies in the sinuses. These eddies increase when the lateral sinus passes into the jugular bulb, which may form a high dome, the longitudinal axis of which forms an acute angle with the longitudinal axis of the lateral sinus.

There may first be a mural conglutination thrombus which adheres to the sinus wall but does not occlude the vessel (Fig. 42). Mural thrombi, according to a widely accepted theory, serve as a defense mechanism against extension of infection to the blood

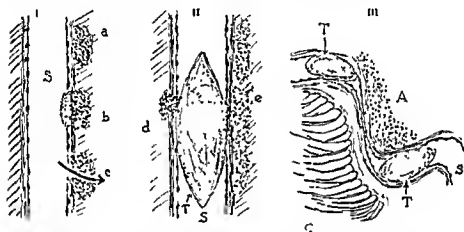


FIG. 42.—Types of thrombophlebitis of sigmoid sinus (S). I a, periphlebitis; b, mural thrombus; c, necrosis of sinus wall, causing hemorrhage from sinus. II: T, obturating thrombus; d, thrombo-endophlebitis; e, periphlebitis. III, compression thrombosis; T, thrombi; A, perisinus abscess, C, cerebellum.

stream and against rupture of the sinus wall. This theory, however, may be disproved by a careful study of these thrombi. A thrombus, although containing no bacteria at the moment of its deposition, may later become the source of septicemia and septic metastases. For this reason it is likely that mural thrombi usually become infected soon after deposition. Likewise, mural thrombi do not in all instances prevent rupture of the sinus wall. In this respect veins and dural sinuses differ conspicuously. Since the sinus walls are not reinforced by a muscular and a definite elastic layer, they are less resistant to pressure from within than are the walls of the veins. In thrombophlebitis the resistance of the sinus wall is even more diminished, owing to the edema and

eventual endarteritis obliterans of the arterioles of the wall. Therefore even the low pressure within the sinus may overcome the resistance of a mural thrombus and may rupture the sinus wall (Fig. 42). This is particularly apt to occur in traumatic sinus thrombosis (p. 152). A hemorrhage from the sinus may be caused by a mural thrombus prior to, as well as after, a mastoid operation. In some cases the hemorrhage does not cause any symptoms and is noted only at operation. In others, there are periodic intense hemorrhages from the external auditory canal. Hemorrhages from the sinus usually indicate severe infection.

EXTENSION OF THE THROMBUS—A thrombus may remain mural and cause septicemia, or it may become organized. Usually, however, it extends in the longitudinal axis of the sinus as well as toward its lumen. Extension toward the lumen, caused by coagulation of blood, changes the mural thrombus into an obliterating thrombus (Fig. 42). It is likely that an obliterating thrombus often forms immediately, without passing through a mural phase. Longitudinal extension of the thrombus is due to coagulation thrombosis, progressive thromboendophlebitis¹ and/or progressive periphlebitis. Therefore in some cases the thrombus extends farther than the periphlebitis, and in others the periphlebitis precedes the thrombus. An obliterating thrombus in the lateral sinus has a spindle shape (Fig. 42).

Primarily, the obliterating thrombus extends in peripheral direction; that is, it extends toward the torcular to the opening of the next tributary vein, so that the blood pouring from this vein into the sinus runs over the tip of the thrombus. Soon, however, the blood peripheral to the thrombus in both the sinus and the tributary vein reverses the flow, and the thrombus then extends toward the heart as well. Formerly, instances of enormous extension of sinus thrombosis were frequently observed. In recent years they have become less common since timely operation usually prevents further extension.

¹In thromboendophlebitis the infection, originating in an infected thrombus, approaches the endothelium of the sinus wall from within (Fig. 42), whereas in periphlebitis the infection extends from the surrounding tissue into the external layer and eventually into the endothelium of the sinus wall.

The thrombus involves particularly the large vessels and does not extend far into the tributary veins. However, the inflammation of the sinus wall extends into the walls of the tributary veins and causes formation of a thrombus which may or may not be continuous with that in the sinus. The sinus thrombus does not always extend continuously, for discontinuous extension may occur, particularly in the superior longitudinal sinus. The pathogenesis of this type is not clearly understood, but there may be an embolic displacement of particles of the thrombus into other parts of the sinus. Occasionally discontinuous extension is simulated by partial liquefaction of one thrombus or by a thrombus which originates in a tributary vein and extends into the lumen of the sinus.

INFECTION BY INFLAMED LYMPH NOOES.—A special type of infection by contiguity is caused by *inflamed lymph nodes adjacent to the veins*, particularly the internal jugular vein. A mural thrombus is usually formed at the site of the node, and occasionally an obliterating thrombus extends from the jugular vein to the lateral sinus. In other instances bacteria originating in the node travel through the wall of the vein and cause septicemia without formation of a grossly visible thrombus.

In a personal case, symptoms of otitic septicemia were simulated by an inflamed node adjacent to the mastoid emissary vein. The lymphadenitis was probably caused by a dermatitis of the scalp.

2. Infection by continuity.—This is due to extension of thrombophlebitis from a small vein into a large sinus. The cavernous sinus is the one most frequently involved, the thrombosis being produced by extension of infection from the ophthalmic veins, carotid plexus, pharyngeal veins and others. Similarly, the lateral sinus becomes involved by extension of thrombophlebitis of the mastoid emissary vein, and the inferior petrosal sinus by extension of thrombophlebitis of the internal auditory vein or vein of the cochlear aqueduct. In all of these instances the thrombus bulges into the sinus, and it is likely that the blood in the sinus drags away particles of the thrombus to cause septicemia and

even metastases before there is actual thrombophlebitis of the sinus. Obviously, infections of this type will cause septicemia more rapidly than infections which extend by contiguity and cause osteitis before they involve the sinus.

OSTEOPHLEBITIS PYEMIA.—All of these instances of infection by continuity demonstrate that otorhinogenic septicemia may occur without thrombophlebitis of one of the large sinuses. However, there are always comparatively large veins, such as the ophthalmic and mastoid emissary veins, which serve as the source of septicemia. In addition, the doctrine has been advanced that venous capillaries which run through the bony plate covering the lateral sinus and carry blood from the mucosa of the mastoid cells to the sinus may become involved during mastoiditis. These small veins are believed to be the source of a septicemia called osteophlebitis pyemia. In my opinion, this concept is open to question from the pathologic point of view. There is no doubt that thrombophlebitis of the anastomosing venules does occur in acute otitis media and mastoiditis. But this thrombophlebitis is more common than the septicemia which is attributed to these vessels. Moreover, the venules are very narrow. That they can harbor enough infective material to cause septicemia seems questionable. For this reason I believe that in these cases there is an additional infection of the sinus wall, because operation usually reveals slight changes such as hyperemia or exudation of fibrin at the wall.

Although the underlying pathologic process is not clearly understood for want of microscopic examinations, osteophlebitis pyemia should be considered a clinical entity, with the following characteristics. (1) It occurs particularly in children and adolescents. (2) It occurs almost exclusively with acute otitis media. (3) It usually occurs in diploic mastoids. (4) It causes metastases in muscles and joints but not in the lungs. (5) At operation, marked hyperemia of the mastoid is noted, but seldom a great amount of pus. (6) The wall of the lateral sinus either is normal or presents a slight amount of fibrinous exudate. (7) The outlook is favorable except in patients debilitated by another disease.

3. *Infection by compression.*—Pressure on the lateral sinus by a perisinus abscess is believed to obliterate the lateral sinus and cause compression thrombosis (Fig. 42). This concept, however, is not in accord with the present view concerning the formation of an infective thrombus. Disregarding the fact that a perisinus abscess rarely compresses the sinus, a thrombus forms only when pressure is associated with an infection of the sinus wall, the infection being of greater importance. Pressure alone is not effective, as shown in cases of large cerebellar abscess which may severely compress the lateral sinus without causing thrombosis.

Compression thrombosis occurs most frequently in children and young people and with both acute and chronic otitis, although more often with cholesteatoma of the tympanic cavity. Operation reveals a large extradural abscess causing conspicuous periphlebitis of the lateral sinus. On incision of the sinus the portion between the knees is seen to be empty or contain a small amount of blood. The cerebral wall of the sinus presents normal endothelium. Toward both the torcular and the jugular bulb the sinus is shut off by obliterating thrombi (Fig. 42). These cases of compression thrombosis should not be confused with cases in which no blood is found in the middle portion of the sinus but there is a fistula in the wall of the sinus and pus escapes from the sinus through the fistula into the mastoid cavity (p. 154).

For two reasons one should be acquainted with the surgical findings in compression thrombosis. (1) If the lateral sinus is carefully incised, layer by layer, and only a small amount of blood escapes, a mural thrombus or a compression thrombosis should be assumed to be the cause. The diagnosis can be made easily by deepening the incision. If blood then gushes from the sinus, it is a mural thrombus; if no more blood escapes, the diagnosis is compression thrombosis. (2) If with a compression thrombosis the sinus is found to be empty, one should not be tempted to incise the cerebral wall of the sinus. This error could eventually cause meningitis or a cerebellar abscess.

In treatment of compression thrombosis, both thrombi blocking free circulation of blood in the sinus should be removed.

Particular attention must be given to the peripheral thrombus blocking the blood flow from the torcular into the lateral sinus. This should be entirely removed until there is a free influx of blood into the sinus. Whether or not the jugular vein should be ligated depends on the further course of the infection.

An infant, aged 15 months, had a large perisinus abscess which had perforated the mastoid cortex near the inferior knee of the lateral sinus. The sinus was covered by a layer of fibrin and granulations 3-4 mm. thick. After removal of this tissue the wall was white and thickened, but smooth. The middle portion of the sinus was incised and found empty. The cerebral wall was smooth and glistening. Although the incision was prolonged to the superior knee of the sinus, no blood was obtained. Then a blunt probe was introduced into the lateral sinus and the peripheral thrombus perforated. Although there was a flow of blood, this technic apparently was not radical enough, because 19 days later the temperature rose to 102.6 F. At this time the child was under the care of a pediatrician who attributed the fever to an upper respiratory infection, although failure of the retroauricular incision to close indicated that pathologic tissue remained in the mastoid. The child was treated for an upper respiratory infection and in a week the temperature fell to normal. In the following five weeks there was occasional fever of 101.5-102.7 F., continuing for one or two days. Two weeks after the mastoid operation, fulminating meningitis developed. At a second operation further extension of the pachymeningitis of the posterior cranial fossa was noted, and although a great amount of thrombus was removed from the lateral sinus, free flow of blood was not obtained from the torcular. Chemotherapy was not available at that time. Death in this case was due to failure to remove the thrombus in its entirety and to postponement of the second operation.

Although the pathogenesis of compression thrombosis is not clearly understood, it is likely that in some instances there is primarily an obliterating thrombus which becomes partially disintegrated. The thrombus remains at the upper knee of the sinus, blocking the blood flow from the torcular, while the rest becomes liquefied and pus escapes by gravity toward the jugular bulb, leaving the middle portion of the sinus empty. In other instances other mechanisms may be at work. At any rate, the sinus is not compressed as long as it contains blood. The middle portion of

the sinus becomes empty first; then the pressure exerted by the perisinus abscess acts on it.

4. *Infection by injury.*—Disregarding trauma to the dural sinuses in head injuries, only injuries due to surgery are to be discussed. Surgical injury most frequently involves the lateral sinus and includes: (1) puncture or incision of the sinus for diagnostic purpose; (2) accidental injury; (3) simple exposure of the sinus. Sinus thrombosis subsequent to surgical exposure of the sinus has particular practical significance which is stressed by the term *traumatic sinus thrombosis*.

Puncture or incision of the sinus wall for diagnosis is followed by rapid closure through formation of a thrombus at the site of injury. There is no infection or further extension of the thrombus. Thrombophlebitis or septicemia following a properly performed incision of the sinus is extremely rare. Accidental injuries of the sinus are more serious. In cases of virulent infection in the mastoid cavity, severe septicemia may ensue. For this reason, injury of the sinus in a case of acute exacerbation of chronic otitis is, other things being equal, more hazardous than in a case of mucous otitis. Accidental injuries are usually caused by bone clips pushed into the sinus wall, making a wound with ragged margins. The margins soon become necrotic, inviting an infection of the sinus wall. Hemorrhage occurs unexpectedly, whereas in incisions for diagnosis the surgeon expects bleeding and is prepared to arrest it. For this reason, the proper technique is not always used in arresting the bleeding and an infection of the injured sinus wall ensues. The hazard is particularly great when accidental injury occurs at the beginning of the operation and the damaged sinus wall remains exposed during the operation. If thrombophlebitis occurs, high fever and chills appear several hours after the operation. The outlook in these cases is not entirely favorable, but timely reoperation and chemotherapy will give a considerable number of recoveries.

TRAUMATIC SINUS THROMBOSIS.—This type has great practical importance because it is the commonest sinus thrombosis due to surgical injury. The sinus is injured only to the extent of simple

exposure of the sinus. Since the wall of a normal sinus is quite resistant to infection, simple exposure is not dangerous (1) if exposure is deliberate, (2) if exposure occurs at the end of the mastoid operation, (3) if the sinus wall is not extremely thin, (4) if the sinus is not exposed within a closed recess of the wound and (5) if no secondary infection of the wound occurs. Among these factors, the last two are the most important. A recess is formed when the sinus is exposed by an incorrect technic (p. 196); when, in a radical mastoid operation, the plastic flap formed from the posterior wall of the external auditory canal covers the exposed sinus, or when, in a mastoid operation, large retrosinus cells are evacuated and the retroauricular incision is primarily closed. In all such instances infective material may be accumulated in the recess created by the operation and cause an infection of the exposed portion of the sinus. Of greater importance is the secondary wound infection which may be followed by a traumatic sinus thrombosis even when the sinus was not exposed during the mastoid operation. The strain of bacteria causing this type of infection has not been studied, but clinical experience indicates that the micro-organisms are usually highly virulent. Postoperative erysipelas originating in a mastoid wound does not often cause an infection of the sinus.

Traumatic sinus thrombosis has the following clinical features. (1) It occurs more often with chronic otitis than with acute otitis. (2) Before the mastoid operation symptoms of sinus thrombosis or septicemia are definitely absent. (3) Immediately after the mastoid operation the temperature is normal or slightly elevated, in contrast with cases of accidental injury of the sinus. (4) There are edema and redness of the skin, and exuberant granulations interfere with normal healing of the retroauricular incision. (5) One to two weeks after the mastoid operation there is a sudden rise of temperature ("alarms fever", *Komplikationszacke*) which ushers in a spiking temperature curve. (6) Operation reveals granulations, necrotic to a great extent, necrosis of the sinus wall and, eventually, a mural thrombus. Owing to the necrotizing inflammation of the sinus wall a large amount of toxins enters the

blood stream before formation of the thrombus. For the same reason the sinus wall frequently yields to the pressure of blood from within, causing sinus bleeding. These findings are at variance with those found with thrombosis following accidental injury of the sinus. In the latter case large thrombi may be found two to three days after the injury, whereas in traumatic sinus thrombosis only a mural thrombus is ordinarily found after one to two weeks. This is probably due to the fact that after injury of the sinus wall the infection immediately involves the entire thickness of the wall and spreads from within outward, as in thromboendophlebitis. In traumatic sinus thrombosis the infection spreads from without inward and causes a necrotizing infection rather than a productive inflammation. (7) Even though the sinus operation is performed, the course frequently is fulminating and downhill, and ligation of the jugular vein and sulfonamide therapy often fail to achieve a cure. (8) If traumatic sinus thrombosis causes metastases they are localized usually in the lungs and rarely in the brain, joints or tendons. The following case may illustrate an infection of this type.

A man, aged 31, experienced pain in the right ear on Sept. 23, 1924, after swimming. On September 27 acute otitis was found on the right side and paracentesis was done. On September 30 the temperature was 101.3 F., and there were a profuse discharge from the right ear and mastoiditis. On October 11 a simple mastoid operation revealed large quantities of pus in the mastoid cells and malar process and granulations in the antrum. There was a perisinus abscess, but the sinus wall was fairly normal except for slight fibrinous deposits. The tegmen was normal. The temperature, which before operation had gradually subsided to normal, rose to 101.3 F. on October 12. The patient complained of pain and there was abundant discharge from the retroauricular incision. On October 14 the temperature was 100 F. The retroauricular discharge continued, but there was none from the external canal. During the night of October 17 there were two chills; temperature in the night was 103.5 F. and in the morning 102.2 F. The dressing was soaked with blood, and the jugular vein was ligated. When the dressing was removed from the mastoid cavity there was a severe hemorrhage from the sinus, and a large tampon was inserted. On October 19 the temperature was 101.3 F.; there were no

chills. On October 20 there was no bleeding from the sinus and there were no chills, but the temperature was 102.7 F. Severe septicemia developed, and the patient died on October 29. At autopsy multiple metastases were found in both lungs. There were parenchymatous degeneration of the internal organs, enlargement of the spleen and universal jaundice, but there was no meningitis or sinus thrombosis.

It is highly important to determine whether or not thrombi found in the lateral or superior longitudinal sinus are infected. Bacteriologic examinations frequently yield different findings according to whether the middle portion or the ends are cultured. In one case the middle portion and in another case the ends may be sterile. Unfortunately, in these examinations the search for anaerobic bacteria has been badly neglected. Since a solid red thrombus may yield a pure culture of streptococci while in putrid portions of the thrombus no aerobic bacteria are found on culture, the surgeon cannot know at operation which portion of a thrombus contains bacteria and which does not. For this reason it is advisable to consider the entire thrombus as infected, because the leaving of an infected thrombus in the sinus involves greater risk than the removal of a sterile thrombus.

If the sinus thrombosis does not cause death, the thrombus may become putrified and/or organized. Sinus thrombosis has a distinct tendency to become organized, particularly mural thrombi and those in small vessels. In the superior longitudinal sinus pre-existing connective tissue septums (p. 14) favor organization of thrombi. The final result is formation within the sinus of granulations which are gradually changed into connective tissue. If there is a proliferation of the sinus endothelium, recanalization of the sinus ensues. In the lateral sinus, occasionally the inflamed wall becomes organized without organization of the thrombus. In these cases the sinus wall is white instead of blue but perfectly smooth, while there is pus or fragments of thrombi within the sinus. This may be observed in cases of both acute and chronic otitis.

If leukocytes originating in the vasa vasorum of the sinus wall invade the infected thrombus, the thrombus disintegrates into

frank pus. The pus either descends by gravity toward the jugular bulb or breaks through the sinus wall, causing a fistula between the lumen of the sinus and the cavity of the mastoid process.

COMPLICATIONS

Thrombophlebitis of the dural sinuses may involve the meninges and brain, or cause metastases and septicemia.

Involvement of meninges and brain.—Since the veins of the

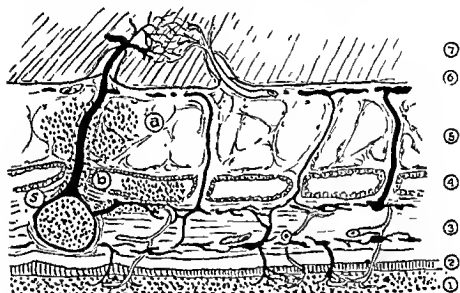


FIG. 43.—Subdural empyema (*b*) and leptomeningitis (*a*) due to thrombophlebitis of a dural sinus (*s*). 1, inflamed mucosa, 2, bone; 3, dura, 4, subdural space; 5, subarachnoid space; 6, pia, 7, brain.

dura, pia-arachnoid and brain drain into the sinuses, thrombophlebitis of a sinus may spread into the dura, pia-arachnoid or brain (Fig. 43). This spread suggests that the venous blood flow is reversed. Such reversal occurs readily in sinus thrombosis because pressure in the veins is low. Extension may occur by continuity or, infrequently, by metastasis. Continuity indicates that the infection spreads from the sinus to, and along, a vein. Metastasis indicates that the infective material is carried by the venous blood from the sinus to a remote part of the brain or meninges.

This material may be an infected embolus or, as suggested by clinical experience, only toxins or attenuated bacteria.

If the infection spreads by continuity, purulent infection results near the thrombophlebitic sinus. For example, if the lateral sinus is the source of infection and the infection encroaches on a vein of the dura, internal pachymeningitis of the posterior cranial fossa will result because the principal veins of the dura run in the internal network (p. 4). If the infection encroaches on a vein of the pia-arachnoid, leptomenigitis will result. The meningitis will be purulent if the veins carry virulent microorganisms and serous if they carry only toxins or attenuated bacteria. If the infection encroaches on a brain vessel, cerebellar edema will occur if the veins carry toxins or attenuated bacteria and a cerebellar abscess if the material is virulent.

Metastatic spread occurs more frequently along the jugular vein than along meningeal and cerebral veins. Disturbances of blood circulation in the brain or hemorrhages into the dura or brain, due to occlusion of the sinuses, occur not infrequently with marasmic thrombi and sinus thrombosis following abdominal operations, but are rare with otorhinogenous thrombophlebitis of the sinuses.

Infections of the brain are caused infrequently by a putrid thrombus which breaks through the sinus wall and involves the brain by contiguity. Occasionally cortical abscesses, but rarely subcortical abscesses, are caused by contiguity (p. 327). A fistula of the sinus wall caused by a putrid thrombus usually drains into the mastoid cavity, not into the brain. From the lateral sinus and jugular bulb the pus may escape through the fistula into the neck, causing abscesses of the neck or retropharyngeal region.

Metastases.—Metastases develop by transportation of bacteria or fragments of a thrombus into other parts of the body. The fragments of a thrombus, called emboli, may be septic or sterile. If a septic embolus or only bacteria are carried away, as usually occurs in otorhinogenous sinus thrombosis, the transportation may be retrograde or direct. Retrograde transportation takes place

when the blood flow in veins draining into the thrombosed sinus is reversed. The septic material may be carried from one sinus to another, for example, from the lateral into the cavernous sinus or from the sinus into the brain or dura. However, reversed blood flow favors the continuous spread of the infection more than retrograde transportation of emboli. The principal mechanism causing metastases is the direct transport of septic material along the jugular vein into the right chamber of the heart and thence into the lungs. Emboli of appreciable size remain in the lungs, causing infarct, abscess or empyema. Lung embolism which is immediately fatal by blocking respiration is rare in sinus thrombosis. If the emboli are small and consist only of bacteria or if there is a patent foramen ovale, the emboli are carried into the major blood circulation and may be deposited in various parts of the body. This may also occur if the emboli originate in a secondary thrombophlebitis of pulmonary veins.

When a septic embolus is arrested, a localized thrombophlebitis develops at the site. The localized thrombus becomes infected, and embolus plus localized thrombus form a single thrombus which ultimately causes a serous or, more frequently, a purulent inflammation of the surrounding tissue. Some writers believe that staphylococci cause metastases more frequently than streptococci and that anaerobic streptococci rarely cause metastases even when a large number are circulating in the blood. It is probable that metastatic abscesses also serve as a source of septicemia.

The most frequent and important metastases are those in the lungs. The diagnosis of an acute lung embolus is often difficult. At onset both physical and x-ray examinations frequently fail to reveal definite findings, and many patients have no subjective complaints. Some complain of pain in the shoulder, believed to be due to a metastasis to the joint, which is actually radiating from irritation of the pleura. Occasionally there are complaints of epigastric pain. In children lung embolism may cause pallor or cyanosis, respiratory movements of the nares and fixation of the thorax with abdominal respiration. Hemoptysis, rusty sputum

and positive results of physical examination of the chest are noted several days later.

SEPTICEMIA

Septicemia occurs when highly virulent bacteria escape continually or periodically from a focus of infection into the circulating blood and when the patient's immunity is inadequate or fails. Some surgeons do not include inadequate immunity in the definition of septicemia, considering the inadequate clearing mechanism of the body the effect rather than the cause of septicemia. For two reasons I feel that the inadequate clearing mechanism of the body should be stressed. (1) If inadequate resistance were the result, and not the cause, of septicemia, the septicemia would always be preceded by simple bacteremia, which does not involve the clearing mechanism of the body. In otorhinologic cases, however, septicemia is seldom preceded by simple bacteremia. (2) The gratifying therapeutic results of building up the patient's resistance in septicemia are better understood when the inadequate clearing mechanism is considered the cause, and not the result, of septicemia. Apparently it is the consensus that in septicemia there is no multiplication of bacteria in the blood, the bacteremia being due exclusively to the invasion of microorganisms which proliferate in the primary or secondary foci of infection.

There is no doubt that thrombophlebitis of a dural sinus may act as a source of septicemia; it is questionable whether bacteria originating in an abscess adjacent to the sinus are likely to migrate through the sinus wall and cause septicemia without *thrombophlebitis of the sinus*. This question particularly concerns the lateral sinus and, to a certain extent, the superior longitudinal sinus. Theoretically it is conceivable that a sinus wall, edematous because of inflammation, allows the migration of bacteria, particularly in acute otitis media of infants and children. In fact, in these cases there is often fever of 104 F. and more which rapidly subsides after simple exposure of the sinus; yet the operation reveals, at most, slight phlebitis character-

ized by a deposit of fibrin on the sinus wall. It is also possible that a distinct anteposition of the sinus facilitates migration of bacteria into the sinus without formation of a thrombus. However, one should keep in mind that the finding of a normal sinus at operation does not prove that there is no thrombus in the jugular bulb or carotid or petrosal sinuses. For this reason, the question can only be answered by microscopic examination of temporal bones. In fact, several cases have been reported of patients who died of otitic septicemia although microscopic examination revealed normal sinuses. However, in most of these cases the carotid sinus was not examined, so the pathology is not clear.

In summary it may be stated that otogenous septicemia without sinus thrombosis probably does exist, although such cases are not common and the pathologic process is not clear.

Symptomatology.—Although every type of septicemia is characterized by a primary focus of infection adjacent to the blood vessels and by the circulation of bacteria in the blood in the presence of diminished resistance, septicemia does not cause uniform symptoms. Both the foci of infection and the invading bacteria vary in many respects. For this reason, the following discussion concerns not septicemia in general but septicemia due to thrombophlebitis of dural sinuses. Also, the term "septic symptoms" applies only to the symptoms of this specific type of septicemia. Discussion of the most important symptoms follows.

SENSORIUM.—The patient's mental state may be altered if highly virulent bacteria circulate in the blood, if the sinus thrombosis is associated with inflammation of the meninges or brain or if septicemia persists a long time. In incipient septicemia, however, the mental state is normal. Distinct euphoria and inclination to make foolish jokes (*moria*) are serious symptoms. In the incipient stage malaise may not be present. It is not rare to have a patient with full-blown sinus thrombosis walk unsupported into a dispensary. The history may even point to a metastasis in one of the joints, although the patient does not seek treatment for several days.

FEVER.—Sinus thrombosis usually causes fever which persists

as long as the immune bodies are capable of fighting the invaders. Where the immune bodies fail, as they do in debilitated individuals and in patients with overwhelming infections, the centers of heat regulation become paralyzed and body temperature does not rise. For this reason, treatment aims not at reduction of fever but at destruction of bacteria, and this is achieved by chemotherapy. Antipyretics should be given only when untoward symptoms attributable to the fever, such as accelerated heart action, dyspnea and dehydration, must be relieved.

A temperature curve characteristic of all cases of septicemia

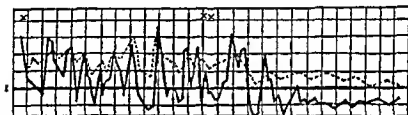


FIG. 41.—Temperature curve for youth, 19, with compression thrombosis following acute otitis. *I*, normal temperature, *x*, simple mastoid and sinus operations and jugular ligation, *xx*, transfusion of 350 cc. of blood

does not exist, but the picket-fence type, namely, the remittent or intermittent type,² is usually noted (Fig. 44). The temperature may fluctuate twice in 24 hours. Occasionally in otitic sinus thrombosis the temperature curve simulates that of malaria. In some cases of nongenous septicemia the temperature curve remains at a consistently high level, especially in infants and children. I believe this type of fever is probably due not to the septicemia but to serous meningitis associated with the sinus thrombosis. No, or only slight, elevation of temperature occurs in sinus thrombosis due to pneumococcus type III or if the sinus is blocked toward the jugular vein by connective tissue. In the latter instance there is apparently no septicemia, although bacteremia and even metastases have been observed. In these cases cure can be achieved by simple exposure of the sinus. There is low fever when

²In remittent fever the temperature rises 1.8 F. or more daily. In the intermittent type it falls to normal, or even below, and then rises abruptly

a sinus thrombus disintegrates and the pus drains through a fistula into the mastoid cavity.

CHILLS.—Spontaneous chills are a symptom of bacteremia, although they do not occur in every case. Immediately after bacterial invasion of the blood stream there is markedly increased activity of the reticulo-endothelial system of the liver, spleen and other abdominal organs to attack the invading bacteria. Since this activity reaches the peak during the chill, it is likely that the chill is caused not by the active bacteria but by their destruction. Therefore blood taken during the chill usually does not contain living bacteria. To obtain a positive culture, the blood should be taken prior to, or at the very onset of, a chill.

With sinus thrombosis the intensity of the chills is highly variable. Frequently there is just a fleeting chilly sensation associated with the chattering of teeth and sweating, or there may be actual shivering. Rarely, the chills are so intense as to be followed by vascular collapse.

PULSE.—The pulse rate usually follows the temperature (Fig. 44). A temperature rise of 1.8 F. is believed to cause a pulse acceleration of eight beats per minute. During a chill and during remission of fever the pulse rate often does not follow the temperature closely; in fact, slight tachycardia may continue even during convalescence. With overwhelming septicemia, septicemia associated with purulent meningitis or cerebellar abscess and, occasionally, even serous meningitis, the temperature and pulse curves may cross, the temperature curve descending and the pulse curve ascending (Fig. 79, p. 357). This has been called "the cross of death." If septicemia causes marked jaundice or if with jugular bulb thrombosis the vagus nerve becomes irritated, bradycardia may be present.

VOMITING.—There may be vomiting between meals but is of little consequence unless it is a frequent occurrence. In this event it is likely that meningitis or cerebellar abscess has set in.

BLOOD.—Hemoglobin content and erythrocyte count are diminished in advanced cases. There are signs of secondary anemia. This, however, is not due to a hemolytic action of streptococci.

because hemolytic streptococci, although hemolytic on the blood-agar plate and in red blood cell suspensions, do not exercise hemolysis in the circulating blood. The number of neutrophils is strikingly increased, while eosinophils are absent. The total leukocyte count may be normal or moderately increased. The finding of toxic leukocytes, characterized by pathologic granules, pyknosis of the nucleus and vacuoles, is generally thought to imply a poor prognosis. In my experience this finding does not allow definite conclusions. Often the proportion of mononuclear to polynuclear neutrophils is increased, showing a shift to the left. In severe septicemia with purulent metastases, myelocytes and myeloblasts may appear. Formerly leukopenia and agranulocytosis were rarely seen with sinus thrombosis, but since the advent of sulfonamide therapy they are noted more frequently. When they appear during therapy the administration of a sulfonamide must be discontinued. Whether penicillin has a similar effect is not yet known. An isolated experience seems to indicate that penicillin may cause fever and leukopenia in children.

The fibrin content is often diminished with severe septicemia, and, probably owing to hepatic damage, the coagulation time may be prolonged. The prolongation is rarely striking, even though hemolytic streptococci do cause fibrinolysis in human blood. With pneumococcic septicemia reduction of coagulation time has been noted. The sedimentation rate is often high. The result of the bacteriologic examination depends on the time the blood is withdrawn, the technic of venipuncture and the technic of preparing the blood culture. These factors explain the variations in number of positive blood cultures reported by different otologists. In addition, a temporary bacteremia does not indicate septicemia unless hemolytic streptococci are discovered in the blood. Even temporary infection of the blood with hemolytic streptococci is not conclusive evidence of septic thrombophlebitis, since the same micro-organisms may be discovered in the blood in the early stage of erysipelas, prior to appearance of the erythema, and in pharyngeal and nasopharyngeal infections. The septicemia of sinus thrombosis is invariably

a mono-infection, even though the aural exudate and thrombus contain multiple strains of bacteria. Blood from the sinus or jugular vein often harbors more bacteria than blood from the cubital vein. Sulfonamides inhibit the growth of bacteria unless para-aminobenzoic acid is added to the culture medium to neutralize the bacteriostatic effect of the sulfonamides in the blood.

SPLEEN.—Engorgement of the spleen is often found with septic sinus thrombosis, although it may be absent in the most severe cases of septicemia.

JAUNDICE.—Generalized jaundice is uncommon with septic sinus thrombosis. Frequently a yellow discoloration of the sclera is seen. This symptom suggests septicemia only in young persons because in old individuals a yellow sclera is often normal.

SKIN.—Petechial hemorrhages in the skin and mucosa, caused by toxic degeneration of the capillaries, is uncommon. This is in contrast with the petechiae found in septicemia with bacterial endocarditis. A skin rash is also rare with septic sinus thrombosis.

Treatment.—This consists primarily of surgical removal of the primary focus, if that is feasible. It is discussed on page 195. If surgery has been performed, or if it is not feasible, conservative measures, including antiseptic and supportive treatment, should be tried.

SULFONAMIDES.—With the demonstration by Domagk in 1935 that prontosil had a specific action against streptococci, a new era of antiseptic treatment was initiated. Many sulfanilamide derivatives and allied compounds have since been studied. Four of these drugs have been successfully employed in otolaryngology: sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine. Sulfanilamide is available in 5 and 7.5 gr. tablets, sulfapyridine in 7.5 gr. tablets, sulfathiazole in 3.85 and 7.7 gr. tablets, and sulfadiazine in 7.7 gr. tablets.

Usually the sulfonamides are administered orally, although they may be given parenterally, using the same total dose. Intrathecal administration has not been shown to give added benefit. Dosage is the same for sulfanilamide, sulfathiazole and sulfadiazine; sulfapyridine, because of its toxicity, is falling into

disfavor. When the infection is severe, dosage based on 1 gr. per pound of body weight, up to 120 gr. in 24 hours, is divided into six parts and a dose given every four hours day and night, orally

ADMINISTRATION AND EFFECTS OF SULFONAMIDES

	SULFANILAMIDE	SULFAPYRIDINE	SULFATHIAZOLE	SULFADIAZINE
Intravenous	No	5% sodium salt in dist. water	1% sodium salt in dist. water	5% sodium salt in dist. water
Subcutaneous	0.8-1% sodium salt in saline	0.3-0.8% sodium salt in saline	0.5% sodium salt in dist. water plus 5% glucose	0.3-0.8% sodium salt in saline
Rectal	1% solution	No	No	No
Peak of blood level after 1 oral dose	4 hr	6 hr	4 hr or less	6 hr
Renal excretion	Rather rapid	Irregular	Very rapid	Slow
Maintaining blood level	Easy	Variable	Difficult	Easy
Acetylation	10-15%	60-75%	0-30%	Slight
Urinalinasis	0	+++	++	+
Cerebrospinal fluid	Present	Present	Scarcely present	Present
Antipyretic	+	+	+	?
Bacteria	Beta hem. streptococcus, meningococcus	Pneumococcus	Staphylococcus, pneumococcus	Streptococcus, pneumococcus, meningococcus, bacteroides (?)
Toxic manifestations	Anorexia, nausea, fever, vomiting, drug rash, leukopenia, agranulocytosis			Rash, injection of sclera and conjunctiva
	Cyanosis, dyspnea, acidosis, acute hemolytic anemia, jaundice, nervous symptoms, chest and abdominal pain, paroxysmal tachycardia, injection of sclera and conjunctiva	Cyanosis, acidosis, injection of sclera and conjunctiva	Acute hemolytic anemia, conjunctivitis, arthritis	

or parenterally or in combination, after an initial dose of twice the amount given in later doses. For intravenous injection, freshly prepared solutions of the sodium salts of sulfapyridine, sulfathiazole or sulfadiazine in sterile distilled water are employed. These ole or sulfadiazine in sterile distilled water are employed. These solutions are strongly alkaline, with a pH ranging from 10 to 11,

and on intravenous injection the sodium ion is promptly split off, leaving the sulfonamide. The injection should be made slowly, at the rate of about 5 cc. per minute or by the slow drip method, and should not be repeated too often because of the danger of thrombus formation. The initial dose is 100 cc. or more of a solution of the sodium salt in distilled water. If intravenous injection is not possible, 500 cc. of a solution of sodium salt in isotonic solution of sodium chloride is given subcutaneously and repeated in six to eight hours, as seems necessary. The highly alkaline drugs may be irritating to the tissues. This dosage results in the immediate establishment of over 10 mg. of free sulfonamide per 100 cc. of blood. To maintain an effective level of free drug, oral therapy should be started immediately after completion of the intravenous injection. If it is necessary to attain a high blood level rapidly, half or all of the entire 24 hour requirement can be given initially by intravenous infusion. Infants, who tolerate the drugs well, are given one-third to one-half the adult dose. Children require from one-half to three-fourths the adult dose.

Usually 5-10 gr. of sodium bicarbonate is administered with each dose to prevent nausea and vomiting due to acidosis. The drug should be administered until the patient is afebrile for at least 72 hours. In cases of meningitis some surgeons continue to administer sulfonamides for three weeks after recovery, in reduced dosage of approximately 60 gr. daily in divided doses. In my opinion this is unnecessary in otorhinogenous complications. Sulfonamides should never be given with sulfates and saline laxatives because this combination may produce sulfhemoglobinemia. *Coal tar derivatives likewise should be withheld.* Patients who are receiving sulfonamides must avoid direct exposure to the sun and ultraviolet light during treatment and for several days after the drug has been discontinued. The drug should not be administered simultaneously with a blood transfusion or the injection of glucose. Fluids should be given parenterally before or after the intravenous injection.

With the exception of sulfapyridine, the sulfonamides, particularly sulfathiazole, are rapidly absorbed and are, in general, uni-

forably distributed. Only bone and fat tissue are refractory. Sulfathiazole does not readily pass into the cerebrospinal fluid and therefore should not be given when there is inflammation of the meninges. All sulfonamides, particularly sulfapyridine, are detoxified to a variable extent by acetylation in the liver and reticulo-endothelial system. The acetyl derivative thus formed is therapeutically inert. Free and acetylated sulfonamides are excreted primarily through the kidneys and, to a lesser degree, into the stomach. Sulfanilamide and sulfadiazine are absorbed and excreted at rates favoring the maintenance of adequate blood levels, with sulfathiazole it may be difficult to maintain the desired blood level.

The most serious objections to the use of sulfanilamide are the eventual damage to erythrocytes, granulocytes and liver and the formation of urinary calculi owing to the comparatively low solubility of the acetylated derivatives. In fact, uremia and agranulocytosis are the commonest fatal complications. With the newer sulfonamide derivatives these dangers, except for the formation of urinary calculi, are reduced. Other toxic reactions to the sulfonamides are listed on page 164.

The following reactions are of particular interest. Cyanosis is common and apparently is due to coloration of the red blood cells by an unknown blue oxidation product of the drug formed spontaneously in the blood, and is not due to the development of methemoglobin or sulfhemoglobin. The hemoglobin function of the blood is not affected during the period of cyanosis. Nausea, vomiting, dizziness, headache and malaise have no significance and, ordinarily, do not require discontinuation of the drug. The slow intravenous injection of a 1 per cent aqueous solution of methylene blue in a dose of 1 mg. per kilogram of body weight is said to relieve the cyanosis in 30 minutes. Fever occurs rarely. If the rise in temperature is only 1-2 degrees, discontinuation of the drug is seldom necessary.

High fever with skin rashes following the first dose usually indicates a natural allergy to a sulfonamide. Acquired allergy is much more common and is frequently responsible for drug fever,

sometimes occurring nine to 12 days after continuous sulfonamide treatment. This "sulfonamide shock" is a warning to discontinue the drug. Otherwise agranulocytosis may ensue, although, in general, there is little evidence to suggest that the minor reactions to sulfonamides are precursors of the more serious reactions. Symptoms subside two to four days after the drug is discontinued. An individual who is allergic to one sulfonamide compound may or may not be allergic to another. A simple test for the determination of allergy is to administer 5 gr. and make hourly temperature readings for at least four to six hours.

The sulfonamides usually reduce the hemoglobin content by 10-20 per cent and leukocytes in the same proportion. Severe granulocytopenia has occurred often without much warning, particularly in addicts to salicylates and other drugs. Acute hemolytic anemia and jaundice indicate that the drug should be discontinued except in desperate cases of septicemia or meningitis, in which the blood should be fortified by repeated transfusions. Erythrocyte and leukocyte counts and determination of hemoglobin content should be made every second day. The urine must be examined for the presence of blood when large doses are being administered. Hematuria is usually due to irritation of the uriniferous tubules by crystals of the conjugated compound. Forced fluids dilute the concentration of crystals in the urine, but since a fluid intake over 2,500 cc. per day may cause too rapid elimination of the drug, it is apparently best to withdraw the drug entirely under these circumstances.

The masking effect of the sulfonamides is believed to be *as important as the toxic reactions*. The drug may reduce fever and headache although septicemia or meningitis is not actually subsiding and may cause headache and fever although septicemia or meningitis is actually decreasing. The masking effect is important but apparently has been overemphasized. Every type of treatment, even surgery, may cause a masking of symptoms, but the disadvantage should be counterbalanced by a close observation of the patient. In the event that a sulfonamide diminishes headache and fever even though a sinus thrombosis or menin-

gitis is advancing, failure of the patient to make proper convalescence, the findings in the cerebrospinal fluid and failure of a retroauricular incision to close give sufficient evidence that the sulfonamide has achieved a symptomatic, but not an actual, cure. On the other hand, fever, headache, nausea and jaundice may be caused by the drug and not by the intracranial complication. In these instances, however, the patient's general appearance is good, the blood examination reveals normal values or leukopenia but not leukocytosis, there are no bacteria in the blood, the pulse rate may fail to follow the rise of temperature, or there are chills although the fever is definitely subsiding. In exceptional cases in which clinical observation does not permit a definite decision, the drug should be withdrawn for one to three days. After withdrawal, toxic symptoms will disappear, while symptoms of septicemia will persist or increase. Only in mucous otitis in old persons are the sulfonamides likely to cause a disastrous masking effect (p. 299), and in some cases of rhinogenous meningitis (p. 308) the masking effect may be confusing.

The disadvantages of sulfonamide therapy are counterbalanced by the bacteriostatic effect. Sulfonamide compounds affect bacteria by interfering with their enzyme system, thus arresting their reproduction. Phagocytosis does not appear to be specifically stimulated. Furthermore, sulfonamides demonstrate a specific action against certain strains of bacteria (p. 164) when distributed in the blood in low concentrations, i.e., the ordinary therapeutic blood levels. In higher concentrations, such as those found in wounds following local application of powdered sulfonamide compound, there is complete inhibition of bacterial multiplication regardless of the drug used or the species of bacteria present. Sulfonamides are not, however, a panacea against streptococci. Certain strains of hemolytic streptococci are refractory, and the anaerobic streptococci are not susceptible to any of the sulfonamides. Pneumococci, particularly type III, differ profoundly in their sensitivity to sulfapyridine. Finally, not all human beings absorb the sulfonamide compounds equally well from the gastro-intestinal tract. Thus failures of treatment occur,

and the number of failures seems to increase in direct proportion to the length of time the sulfonamides are in practical use. Perhaps their widespread employment has caused a "survival of the fittest" among micro-organisms.

The effect of sulfonamide treatment of septic sinus thrombosis is not thoroughly understood. The consensus seems to be that sulfonamides do not act directly on the thrombus because there are no blood vessels to carry the drug into the thrombus. Furthermore, the thrombus may contain, or may be surrounded by, pus and necrotic tissue which interferes with the proper action of the drug. As to the blood stream infection, the bacteria frequently invade the blood intermittently, so that the drug can act only if the blood level is at its peak at the moment of the bacterial invasion. Moreover, in this action the sulfonamides are only an ally, however potent, of the more powerful defense mechanism of the body. Therefore the primary effect of sulfonamide therapy in septic sinus thrombosis is apparently to prevent the spread of infection within the sinus wall, dura and leptomeninges. The "antispreading effect" is of the utmost importance. Even the most radical operation for septic sinus thrombosis removes only the thrombus, leaving the inflamed sinus wall. Since almost all post-operative complications originate in the inflamed wall, definite advances would be made if sulfonamide therapy actually arrested further progress of the inflammation in all instances. In general, however, the effectiveness of sulfonamides in septic sinus thrombosis is not as spectacular as in meningitis. Perhaps more could be achieved if in septicemia the same large doses could be administered as in meningitis, but this is not feasible. In septicemia the dose must be carefully adjusted to the patient's condition because both the septicemia and the sulfonamides may have a deleterious effect on the hemopoietic system, whereas meningitis does not depress the hemopoietic system to the same degree. In fact, experience indicates that leukopenia occurs more frequently in septic thrombophlebitis than in meningitis treated with sulfonamides, even though larger doses are given in meningitis than in sinus thrombosis.

PENICILLIN.—To avoid the disadvantages of sulfonamides, antibiotic² agents, especially penicillin and tyrothricin, have been developed. Penicillin is produced by the growth of certain strains of *Penicillium notatum* on suitable liquid cultures. It is active mainly against gram-positive cocci and is almost entirely uninfluenced by the number of bacteria to be inhibited. Penicillin acts only on certain bacteria, indeed only on certain strains of the sensitive bacteria, and has no effect on the growth of others. Several strains of pathogenic, saprophytic and commensal bacteria are thought to produce in culture a substance called penicillinase which is capable of inactivating penicillin in vitro. Sensitive microorganisms may develop a resistance to penicillin owing either to a small initial dosage or to administration of large doses followed by an interval of no therapy, during which the blood penicillin content becomes therapeutically negligible. Streptococci are more resistant than pneumococci, and staphylococci, of all the gram-positive organisms, are the most resistant to both penicillin and tyrothricin. A strain may be sensitive to penicillin and resistant to tyrothricin, and vice versa.

In otolaryngologic practice, penicillin is indicated in staphylococcus infections with or without bacteremia, hemolytic streptococcus infections with bacteremia and serious local infections, anaerobic streptococcus infections and pneumococcal infections of the meninges. It is contraindicated in all gram-negative bacillary infections (*Bacillus proteus*, *B. pyocyaneus*, *Escherichia coli*, *B. Friedlaender*, *Haemophilus influenzae*). Penicillin is unable to penetrate necrotic tissue and walled-off abscesses.

The purified extract of penicillin is active in very high dilutions, and its action is not inhibited to any appreciable degree by purulent exudates, tissue cells or para-aminobenzoic acid—substances which nullify the bacteriostatic action of the sulfonamides. It is not toxic, even in large doses. It is bactericidal in the lower dilutions and bacteriostatic in higher ones. It acts much more slowly than tyrothricin but shows definite killing power in

²Antibiotics may be defined as "antimicrobial agents produced by living bacteria, yeasts, molds and other plants" (Kolmer).

two hours and bacteriostasis during the two hours before the killing effect becomes marked. No drug which may be administered systemically, including the sulfonamides, is known to interfere with the action of penicillin, nor is any drug known to be contraindicated with penicillin. Sensitivity and sensitization to penicillin are uncommon, but occasionally thrombophlebitis at the site of continuous intravenous injection, urticaria, conjunctivitis and chills, with or without fever, have been observed after intravenous injections.

The potency of the various penicillin dilutions may be approximately calculated in terms of Oxford units.⁴ An Oxford unit is the amount of penicillin that will inhibit the growth of a standard inoculum of *Staphylococcus aureus* in 50 cc. of broth. This is a variable measure of potency because many strains of staphylococci are resistant to penicillin, and the strains that are sensitive vary considerably in degree of sensitivity. Penicillin is supplied in vials of 100,000 units each. It is extremely soluble so may be dissolved in sterile isotonic solution of sodium chloride or 5 per cent dextrose solution (2,500–5,000 units per cc.). Penicillin in solution has only a limited stability which seldom exceeds a period of one week.

Penicillin is absorbed from the intestinal tract, but oral administration is not satisfactory because of the destructive action of the gastric juice. Because it is rapidly excreted by the kidneys and from the liver by way of the bile, large doses must be given at frequent intervals to maintain an effective blood level. Intramuscular injection is the most practical method of administration, although it may be given intravenously or intrathecally, can be injected into abscess cavities and may be applied locally without damage if the preparation is pure. Intravenous administration gives almost immediate high initial concentration of penicillin in the blood plasma, followed by an abrupt fall one-half to three hours after injection. A dose of 10,000 units injected

⁴The International Conference on Penicillin adopted an international unit of penicillin defined as the specific penicillin activity contained in 0.6 microgram of the international penicillin working standard. This unit is approximately equivalent to the Oxford unit.

intravenously gives a more than adequate blood concentration immediately and a less than adequate concentration about 30 minutes later. Intramuscular administration is followed by less rapid absorption and provides a concentration in the blood which does not reach as high a level as with intravenous injection but may last for three hours. Subcutaneous injections act similarly to intramuscular injections but are likely to be painful. It is preferable to give large doses initially because some organisms may build up resistance to penicillin. When multiple intravenous or intramuscular injections are given, the intervals between injections should not exceed three hours. Otherwise the rapidity with which penicillin is excreted causes such wide variations in blood concentration that optimal results can scarcely be expected. For the same reason it is advantageous, when a large daily dosage is indicated, to shorten the interval between injections rather than to increase the amount of each dose.

In severe acute infections associated with septicemia, 100,000-300,000 units should be given for the first three days. This amount should be administered intravenously either by injecting 10,000-15,000 units in 1 cc. of saline every two hours or by the drip method. If the latter method is used an initial dose of 200 cc. of a solution containing 40,000 units in 1,000 cc. of saline solution or 5 per cent glucose solution is administered at a fairly rapid rate. Thereafter the rate of administration is regulated at 30-40 drops per minute. This method provides a blood concentration of at least 0.2 units per cubic centimeter. With improvement, treatment may be continued by intramuscular injections of 10,000-30,000 units dissolved in 2-4 cc of isotonic salt solution every two to three hours. The dosage for children is 5,000 units every two hours. As the patient improves the dose may be reduced, the three hour interval being maintained. The dosage in children is usually 40,000-50,000 units per day, depending on the nature of the infection and the underlying pathologic process. These figures are not based on hard and fast rules. Individualization of treatment is necessary. Some staphylococci are killed by a blood concentration of 1/50 unit per cubic centimeter, whereas

other strains may require 2 or more units. For this reason some patients may recover from serious infections following the administration of 40,000-50,000 units in 24 hours, while others require 200,000 or more units in the same period. Since it is difficult at present to define adequate blood concentrations, it is better to err on the side of excessive dosage rather than to risk the use of insufficient amounts.

The temperature curve is not a perfect criterion of the clinical progress, but bacteriologic examination, diminution of pain and improvement of patient's appetite and general condition indicate the effect of treatment. In some cases of severe infection penicillin should be continued for about seven days after the temperature has reached a normal level, the total dose in 24 hours being reduced by half. The hemoglobin content and red cell count usually rise during treatment. The white cell count may drop as the infection is controlled, but actual leukopenia does not result.

TYROTHRICIN.—This antibiotic is isolated from peptone cultures of the aerobic soil bacterium *Bacillus brevis*. From tyrothricin two crystalline compounds, gramicidin and tyrocidine, have been separated. Gramicidin is bactericidal for gram-positive organisms only; tyrocidine contributes to the solubility and stability of the active principle gramicidin. Tyrothricin has a strong hemolytic action and should not be given intravenously or subcutaneously. In direct contact it kills gram-positive aerobic and anaerobic bacteria with greater rapidity than penicillin. Penicillin, however, penetrates the tissues and reaches bacteria which would not be killed by tyrothricin. The latter can be used only for local application to surgical wounds and infected mucous membranes.

Immune serum is effective in pneumococcus type III infections and can be given with penicillin. The administration of vaccines in septicemia is apparently useless because septicemia is not caused by a multiplication of bacteria in the blood; the micro-organisms, originating in a focus of infection, invade the blood stream where they are killed and thus cause autovaccination

of the body. The administration of Pregel's solution, mercurochrome, metaphen, trypanflavine and a host of other antiseptics is replaced by modern chemotherapy.

This discussion does not advocate hard and fast rules concerning modern chemotherapy. Some patients require extensive chemotherapy, and others recover with minimal doses. Osteophlebitis pyemia in a child has, from the onset, a more favorable prognosis than sinus thrombosis in an arteriosclerotic patient. Mucosis otitis in an incompletely pneumatized temporal bone of a child does not threaten with the great danger of meningitis as does mucosis otitis in an extremely pneumatized temporal bone of an old person. In all such cases the chemotherapy must be adjusted to the underlying pathology. Furthermore, if the surgeon is certain that he has eliminated the entire focus of infection, the intensity of postoperative chemotherapy may be more readily reduced than when surgery was not radical. Finally, the bone marrow responds differently to sulfonamides in individual patients. For this reason, the use of chemotherapy should not be an automatic procedure. Nor should the dosage be based exclusively on bacteriologic findings. Clinical examination and evaluation of the underlying pathology are of the utmost importance in order to achieve intelligent administration of chemotherapy.

SUPPORTIVE MEASURES.—Owing to the spectacular success of chemotherapy, supportive treatment, which has the purpose of increasing the fighting forces of the body, is frequently considered to be outmoded in cases of septicemia. This underestimation is by no means justified since the favorable results of the treatment of otitic sinus thrombosis prior to the advent of chemotherapy were, to a great extent, due to the supportive treatment. This type of treatment still plays an important part in the care of septicemia.

Maintenance of the patient in as nearly a normal nutritional state as possible is imperative. Food rich in calories and easily digestible is required. If possible, 15-20 calories per pound of body weight should be given daily. Patients frequently complain

of anorexia, so food should be served in small quantities, independent of mealtime, and close attention should be given to the regulation of bowel movements. Vitamin supplements are valuable.

Of equal importance is the fluid intake, which combats dehydration, acidosis and alkalosis. The symptoms of dehydration are parched mouth, marginal redness of the tongue, dryness and darkness of the dorsum of the tongue, sunken eyes and dry inelastic skin. The degree of dehydration can be estimated by determination of the plasma volume and plasma protein concentration. Alkalosis ensues if sufficient chloride ions are lost with gastric juice by vomiting. In these cases a sharp reduction in the amount of chlorides excreted in the urine² is the first measurable reaction of the body to chloride deficiency. In acidosis an excess of acid ions is produced by faulty metabolism of fats. The H-ions neutralize the sodium ions and other buffer solutions of blood and tissues, so that the amount of buffer substances available is reduced. In patients with high fever, acidosis may be caused by the production of lactic acid. With acidosis there is low carbon dioxide-combining power of the plasma. The test for combining power actually measures the amount of buffer substances in the blood and therefore gives information regarding the alkali reserve.

To combat these conditions fluids must be given. If the fluid contains both chlorides and glucose, protection against both acidosis and alkalosis is assured. But since the administration of fluids may promote a brain edema and increase intracranial pressure, *no arbitrary rules can be given concerning the fluid intake in intracranial complications.* In adults a relatively larger amount of fluids can be permitted than in children, who are more likely to respond with brain edema. To a patient who is dehydrated by severe septicemia but does not show signs of intracranial hypertension, more fluids can be given than to a patient who is dehydrated by an acute brain abscess and does present symptoms of intracranial hypertension. For these reasons

²The normal output of chlorides in the urine is 5-10 Gm.

individualization of administration of fluids is imperative in intracranial complications.

In general, the following guide may be of assistance. (1) Oral intake of fluids is preferable to parenteral administration except in somnolent patients who lost a great deal of blood at operation. (2) When dehydration is associated with intracranial hypertension attention should be directed toward intracranial hypertension first and then to dehydration. (3) The large amount of fluids frequently required in abdominal surgery to combat severe dehydration is almost never required in intracranial complications. (4) Saline solutions should not be given because eventual retention of sodium chloride encourages the development of brain edema.

To combat dehydration by parenteral administration, solutions of glucose in distilled water or blood transfusions should be given. The concentration of glucose solutions should not exceed 5 per cent, if administered subcutaneously. By the continuous drip method the concentration should not exceed 10 per cent. The solution should be given slowly at a rate of about 200 cc. per hour, because rapid administration causes marked diuresis, which increases dehydration. To combat intracranial hypertension, 100-150 cc. of a 50 per cent dextrose or sucrose solution may be given intravenously as often as necessary to keep the pressure within reasonable limits. Hypertonic solutions are believed to reduce intracranial pressure about 50 mm. for about one hour. After this reduction the pressure returns slowly to its original, or slightly higher, level. The same purpose is served by administration of magnesium sulfate and regulation of the fluid intake. Magnesium sulfate may be given orally (2 oz. in 4 oz. of water every morning to adults) or by the drip method per rectum. If there is moderate dehydration but no actual or impending intracranial hypertension, one should calculate 1,500 cc. of fluid for urine, 2,000 cc. of fluid for respiration and a numerical replacement of fluid lost in vomiting and hemorrhage. All of this amount, or the greater part of it, can be given by mouth. If with this fluid intake the urinary output

is 1,500 cc. daily and specific gravity of urine 1.015, one may assume that fluid intake and electrolyte balance are adequate.

If there is moderate dehydration associated with intracranial hypertension the calculation is the same, but the administration of fluids is different and total fluid intake is reduced to the minimum required. Only 1,000-2,000 cc. of fluids is given by mouth in 24 hours. The rest is administered parenterally, 500-1,000 cc. of hypertonic dextrose or sucrose solution being given intravenously. In addition, 200-400 cc. of a 50 per cent solution of magnesium sulfate is given by the drip method per rectum. Obviously the amount of fluids given depends not only on the degree of dehydration and intracranial pressure but also on the condition of the kidneys and heart, which are frequently damaged in septicemia.

For blood transfusion, the following preparations are suggested: (1) whole unmodified blood (direct transfusion); (2) citrated or modified blood (indirect transfusion), (3) specifically immunized blood (which can be used only in cases of protracted septicemia); (4) nonspecifically immunized blood; (5) immunized blood and blood serum from convalescent donors. A present, indirect transfusion (8 cc. of a 5 per cent sodium citrate solution to 100 cc. of blood) is usually employed, although malaise and chills are regarded as being more common with this than with direct blood transfusion. To maintain the blood sugar level dextrose can be added to the sodium citrate solution as indicated. Because of the rapid disintegration of leukocytes and the deterioration of complement and bactericidal activity, stored blood should not be used in septicemia.

The purpose of blood transfusion is to replace erythrocytes lost in anemia, to restore the level of plasma proteins in hypoproteinemia, to increase the blood volume, to regulate the water balance, thereby diluting toxic blood, to increase coagulability of blood, to add immunologic factors and, eventually, to stimulate hemopoiesis. The last-mentioned effect may counteract, to some extent, the unfavorable effect of the sulfonamides on erythropoiesis and production of natural immune bodies.

In the average adult weighing 155 lb. one may expect a rise of approximately 10 per cent in hemoglobin value for each 500 cc. of fresh whole blood transfused, an increase of red cell count of approximately 100,000 per cc. for each 100 cc. of blood transfused and a rise of platelets of 20,000-40,000 per cc. for each 400 cc. of blood transfused. Small transfusions, in adults of 100-200 cc. (never over 500 cc.) at two to three day intervals and in children and infants 1-1.5 cc. of whole blood per pound of body weight, are more efficacious than one transfusion of a large amount of blood. It has been emphasized that individuals who have repeated blood transfusions from a particular donor occasionally have hemolytic transfusion reactions even when the donor's and the patient's blood groups are compatible. These are due to an irregular isoagglutinin called the Rh factor.

Blood transfusions are given before, during and after surgical operations. Because in dehydrated patients the loss of a small amount of blood may assume an importance out of all proportion to the amount of blood lost, blood transfusions should be given such patients prior to the operation. Blood is given by the continuous drip method during operations which are notoriously hemorrhagic. I have used this technique successfully in cases of osteomyelitis of the skull and extensive sinus thrombosis. After the operation blood transfusions should be given when, despite a radical operation, septic fever continues or anemia is increasing and the general condition poor. Transfusion should not be postponed until death is impending, because in this phase the transfusion is, as a rule, useless.

Whatever one may think of the value of transfusion, it certainly does not harm the septic patient. Brain edema and vascular collapse, which may occur in meningitis or brain abscess after a blood transfusion, are rare in septicemia. Embolism due to transfusion is also infrequent. In fact, most adverse post-transfusion reactions are referable to (1) the use of specifically incompatible blood, causing agglutination of the donor's cells by the recipient's serum, and (2) presence in the donor's blood serum of nonspecific protein components or food proteins which, when transfused,

cause reactions suggesting allergic phenomena such as slight urticaria and moderate angioneurotic edema. The transfusing of incompatible blood^a causes agglutination and hemolysis of the red blood cells which, in turn, cause a blocking of the renal tubules with blood pigment, leukocytes and desquamated cells. In most cases uremia, coma and death follow in four to 15 days after the transfusion. Treatment in such cases is seldom successful. With a severe anaphylactic or proteolytic reaction to the transfusion there are light fever, asthma, involuntary evacuation and death in a few hours. Anaphylactic shock is infrequent, particularly in patients with septicemia. The milder types of allergic reactions, characterized by urticaria, slight angioneurotic edema, eosinophilia, difficulty in respiration with asthmatic rales and involuntary evacuation, can almost invariably be relieved by the subcutaneous injection of 0.3-1 cc. of 1:1,000 epinephrine.

In 5-10 per cent of patients with intracranial complications, in those with brain abscess more frequently than in those with septicemia, a properly performed transfusion causes chills or rigor followed by a rise of temperature to 105 F. and more. This usually occurs within 15 minutes to one hour after the transfusion and continues for 15-30 minutes. In favorable cases the temperature returns to normal or to the pretransfusion level in one to three days. Persistent fever is due to the septicemia, and the outlook is not favorable. Some surgeons report that progressive sinus thrombosis may be masked by transfusion. I have never noted a masking effect.

^aThe determination of compatibility is performed with the serum of the blood groups A and B. The test may yield four different results. (1) Neither serum causes agglutination of the recipient's erythrocytes. A patient of this type belongs to group O. (2) Only the serum of group B causes agglutination. A patient of this type belongs to group A. (3) Only the serum of group A causes agglutination. A patient of this type belongs to group B. (4) Both serums cause agglutination. A patient of this type belongs to group AB. Donor and recipient must belong to the same group. Even universal blood, group O, should be used only for patients belonging to group O and not for patients belonging to one of the other three groups, except in emergencies. Even if the donor's blood is of the same type as that of the patient, it should not be used until a direct compatibility test has been carried out which demonstrates that the cells of the donor can be mixed with the serum of the recipient and the cells of the recipient with the serum of the donor without occurrence of either hemolysis or agglutination.

In the presence of pulmonary edema, severe nephritis and serious myocardial degeneration transfusion should not be performed. In senile patients and in the presence of asthenia and brain abscess it should be performed at an extremely slow rate and only by trained technicians. Administration of penicillin is no contraindication to blood transfusion; sulfonamide therapy is not compatible with blood transfusion.

Blood substitutes (plasma) are not routinely employed in septic sinus thrombosis. Alcohol, frequently used in the past, is now rarely employed. Alcohol has neither an antibacterial nor an antitoxic effect. On the contrary, there is evidence that it reduces resistance to infections. Nevertheless, I administer small doses of alcohol by the oral, never by the intravenous, route because alcohol reinforces the action of the heart and replaces a certain amount of fat and carbohydrates in the metabolism. Patients with septicemia usually tolerate large amounts of alcohol.

Heparin has been suggested to prevent agglutination of platelets to form white thrombi and to interfere with the union of prothrombin and calcium to form thrombin. One thousand units (10 mg. of sodium salt of heparin) of heparin and 5 mg. of chlorobutanol are diluted in 100 cc. of physiologic saline solution. The solution is given intravenously until the coagulation time is between 20 and 40 minutes. About 25 drops should be given per minute and the treatment may be continued for 11 days. By the continuous drip method, 15 cc. of heparin added to 1,000 cc. of physiologic saline solution is given at the rate of 20-40 drops per minute. Even more efficacious in prolonging the coagulation time is coumarin. It has the advantage of a more prolonged action and can be taken by mouth. Another anti-thrombotic preparation is germanin, given in a single dose of 0.5-1 cc. intravenously. Slight albuminuria occasionally occurs. The efficacy of these preparations in septic sinus thrombosis is not yet definitely determined. In thrombophlebitis of the cavernous sinus they are apparently useful. However, heparin is not without danger, since large cerebral hemorrhages and hematuria have been noted after its administration. The administration of

reduced iron in septic anemia is not of great value. Nevertheless, doses of 0.5 Gm. three times a day can be given orally if this does not interfere with the patient's appetite.

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THROMBOPHILEBITIS OF THE LATERAL SINUS PATHOLOGY

Of the dural sinuses surrounding the temporal bone, the lateral sinus is most frequently involved owing to its prominent position in the mastoid process and the numerous veins running through its bony plate which establish an anastomosis between the mucosa of the mastoid cells and the sinus. A sinus which bulges into the mastoid antrum is no more exposed to infections than a sinus 2-3 cm. from the antrum. On the contrary, infection of a well pneumatized mastoid process involves the sinus more frequently than does a localized infection of the mastoid antrum with an adjacent sinus. This may be one reason for the comparatively low incidence of septic sinus thrombosis in infants and children, in whom mastoid pneumatization is incomplete and the sinus lies near the antrum. The right lateral sinus protrudes deeper into the mastoid process and is said to become involved

more often than the left sinus. Many surgeons, however, including myself, find no striking difference between the two sides. More important than the structure of the mastoid is retention of pus in the antrum and mastoid. In this respect there is no clear difference between acute otitis and acute exacerbation of chronic otitis as far as frequency of sinus thrombosis is concerned.

Infection of the lateral sinus occurs by contiguity, by continuity, by compression and by injury. It involves primarily the area of the superior knee of the sinus and may extend peripherally toward the torcular or centrally toward the jugular bulb. Occasionally thrombophlebitis of the lateral sinus occurs if an infection of the pharyngomaxillary space reaches the jugular vein or if the jugular vein becomes infected from adjacent lymph nodes, in which case the thrombophlebitis of the jugular vein may extend to the lateral sinus.

COMPLICATIONS

Regardless of whether the thrombus is mural or obliterating, parts of it may be carried into the blood stream and cause metastases. The emboli may originate in the central end of the thrombus and be carried through the jugular vein into the right heart, or they may originate in the peripheral end of the thrombus and be carried by retrograde transportation into the vertebral plexus or jugular vein of the opposite side. In either instance the metastases can be divided into those to (1) the meninges and/or brain, (2) the thorax, (3) the abdomen and (4) the joints, muscles and skin. Metastases to the larynx, eye, thyroid gland and spine are rare. Some surgeons hold that the distribution of metastases depends on the selective affinity of the micro-organisms for certain organs, but there is not much evidence to support this concept.

Metastatic meningitis and brain abscess are rare. Pulmonary infarct and lung abscess are the most common metastases, regardless of whether the sinus thrombosis is due to acute or to chronic otitis. In fatal sinus thrombosis due to chronic otitis metastases are nearly always found in the lungs at autopsy. The

abscesses are usually close to the surface of the lungs, involving the pleura. Prognosis must be guarded. If there is only a single metastasis and if the diagnosis is made sufficiently early, recovery often follows chemotherapy. Metastases to the abdomen (abscesses of the spleen, kidney, liver, genital organs) are rare, but parenchymatous degeneration of intestinal organs is often found in fatal cases. The outlook is poor because the diagnosis is rarely made early. Metastases to the muscles and joints occur particularly with acute otitis. The infection is localized primarily around the joints, especially in the tendon sheaths. Later it may invade the joint itself, in which case ankylosis always ensues. I once noted metastatic osteomyelitis of the femur in a case of sinus thrombosis associated with Banti's disease. The prognosis for life with these metastases is not bad. Metastases to the skin are dangerous if they cause Fraenkel's dermatomyositis, which consists of hemorrhagic and septic edema of the muscles, without abscess formation, and a skin infection resembling erysipelas. I have seen a number of cases presenting metastatic dermatomyositis of the lateral part of the neck below the retroauricular incision, all fatal. Chemotherapy will probably improve the outlook for these complications. The dermatomyositis must not be confused with simple erysipelas. In erysipelas there is a picket-fence type of fever and the patient's general condition is not strikingly altered, whereas in dermatomyositis there is continuous high fever and the patient is acutely ill.

Next to metastases, the commonest complication of lateral sinus thrombosis is extension to the dura, leptomeninges or brain, causing internal pachymeningitis, leptomeningitis or a brain abscess. In fact, among fatal cases of lateral sinus thrombosis over 50 per cent had meningitis or brain abscess. The pathogenesis of these complications is discussed on page 155.

SYMPTOMATOLOGY

In some cases of sinus thrombosis the diagnosis can be made at first glance, while in others it is exceedingly difficult. Owing to the progress in otolaryngology, advanced cases of septic sinus

thrombosis are seen less frequently than in the past. Nevertheless they still occur because diagnosis is not made early in the initial phase of sinus thrombosis. For this reason, the following paragraphs stress the symptoms of incipient sinus thrombosis.

Systemic symptoms.—The patient's general condition is markedly impaired if there is overwhelming septicemia; if the thrombosis causes additional complications of the meninges or brain or if the septicemia continues for a long time. But it must be strongly emphasized that patients with incipient septicemia frequently feel perfectly well and have not the slightest idea that they are dangerously ill.

In advanced cases the temperature is of the picket-fence type (Fig. 44, p. 160) or, especially in children, is markedly elevated without remissions or intermissions. In certain cases (p. 160) the fever is inconspicuous or even absent. In these cases the prognosis is frequently good and recovery may follow a simple exposure of the sinus. However, if the sinus is not surgically exposed or no operation on the mastoid is performed, the course may be protracted over three or four months, during which the temperature is normal or slightly raised, occasionally interrupted by sudden fever and chills one to three weeks apart. In all this time the sinus infection gradually advances until, immediately before death, the picket-fence type of temperature becomes evident.

The temperature has practical significance in incipient sinus thrombosis. Acute otitis in adults causes fever only during the first few days, when it is due to a common cold, influenza or some other primary infection. Nor does chronic otitis in adults cause fever, even with an acute exacerbation. Therefore, if in acute otitis or in chronic otitis without acute exacerbation there is fever over 102 F. and it continues for a few days, one must suspect an infection of the lateral sinus unless there is a definite intercurrent infection of the lungs, tonsils, appendix or other organs. When the fever is caused by the ear infection, the sinus should deliberately be exposed if an operation is performed. Obviously, in a case of this type extensive sinus thrombosis will

not be found. Often there is only periphlebitis. Nevertheless, this finding justifies the procedure.

If the patient has been treated with sulfonamides an elevation of temperature is even more indicative of infection of the sinus wall. Here the masking effect of sulfonamides is believed to be evident. Undoubtedly, a sinus thrombosis may progress despite sulfonamide therapy. Nevertheless, correct diagnosis should not be too difficult, for several symptoms cannot be masked by sulfonamides, indicate a severe type of otitis and call for x-ray examination of the mastoid. These include sagging of the superior posterior wall of the external canal, abundant aural discharge, mastoid symptoms, obstinate acute exacerbation of chronic otitis, headache and sleeplessness. If these symptoms do not permit the diagnosis of sinus thrombosis, at least they indicate operation on the mastoid.

The importance of chills in incipient sinus thrombosis is controversial. In fact, an actual rigor is not common; but if fleeting chilly sensations are considered as rudimentary chill and if leading questions are asked in taking the history, the finding of chills is common with incipient sinus thrombosis in adults. In children chills are of minor importance.

The pulse rate has no characteristic features in incipient cases. Vomiting occurs only in advanced cases, and even then is not marked. The blood examination yields important data in advanced cases (p. 162). In incipient cases the blood changes are less conspicuous, particularly if the patient has been treated with sulfonamides. If bacteria are discovered in the blood, they are hemolytic streptococci in 60-90 per cent of cases; pneumococcus type III is found occasionally, and *Bacillus proteus vulgaris*, *B. coli*, *B. pyocyaneus* and Friedländer's bacillus are found exceptionally in sinus thrombosis due to chronic otitis. Proper attention has not been given the cultivation of anaerobic bacteria, particularly in cases of cholesteatoma of the tympanic cavity, although various anaerobic strains have been reported to have caused septicemia, among them *B. ramosus*, *B. perfringens*, *Micrococcus foetidus*, *Staphylococcus parvulus*, *Spirillum nigrum*,

B. serpens and *Vibrio funduliformis*, some of which are gas-producing.

General brain symptoms.—Headache is not severe in sinus thrombosis unless a meningeal infection is also present. In fact, headache is often absent. If present, it is localized in the occiput or forehead. Occasionally patients complain of sharp pain shooting through the skull. Eyeground changes are not uncommon, they usually consist only of passive hyperemia and tortuosities of the retinal vessels. In 10-16 per cent of cases papilledema is seen. It may occur in either obliterating or mural thrombosis, but it may also appear several weeks after the operation, particularly if the jugular vein was ligated. In any case, the papilledema does not seriously impair the outlook unless it is gradually increasing. In some cases of obliterating sinus thrombosis passive hyperemia of the retinal veins can be produced by pressure on the jugular vein of the opposite side—the sign of Beck-Crowe. This sign is of minor practical importance, although it is more reliable than the sign of Dishock, which is believed to indicate sinus thrombosis when pressure on the jugular vein fails to cause narrowing of the inferior nasal meatus. In bilateral sinus thrombosis in children the entire head may become edematous and the veins of the scalp strikingly dilated.

The cerebrospinal fluid is normal in the initial stages of sinus thrombosis. Pressure may be slightly increased. Some authors report the finding of bacteria in clear fluid. I have never had this experience. In advanced cases the cerebrospinal fluid may become cloudy, but remains sterile if the thrombophlebitis spreads far within the sinuses or causes internal pachymeningitis of the posterior cranial fossa. The Tobey-Ayer test is discussed on page 50. Recently it was suggested that the internal jugular vein be punctured below the tip of the mastoid process to measure the venous pressure during and after compression of the vein. In the absence of lateral sinus thrombosis there is a significant rise and fall of venous pressure, whereas in sinus thrombosis, obliterative or partial, there is either no change or a slight rise and fall. I have not used this test.

Focal brain symptoms.—Focal symptoms are not caused by sinus thrombosis; but metastases to the brain, hemorrhages in the brain, serous meningitis, thrombosis of the pial veins or erroneous puncture of the brain in the presence of a sinus thrombosis may cause focal symptoms such as aphasia, Babinski's sign, ankle clonus, jacksonian attacks and hypotonia of the extremities.

Ear symptoms.—Of all the symptoms of lateral sinus thrombosis, the systemic and aural symptoms are the most important

*ACUTE OTITIS.*⁷—The most suggestive finding is surgical mastoiditis. There are moderate discharge, a hyperemic drum membrane, occasionally presenting a nipple, a sagging of the superior and posterior walls of the external canal and edema and tenderness at the tip of the mastoid. (Griesinger's sign, consisting of localized tender edema at the posterior boundary of the mastoid process, is often absent in sinus thrombosis, and often present in simple mastoiditis.) These ear symptoms indicate mastoiditis, not sinus thrombosis. However, their association with the systemic symptoms previously mentioned suggests sinus involvement and the sinus wall should be inspected at operation.

Frequently the discharge fluctuates, being abundant one day and scanty the next. The periodicity may be caused by temporary obstruction of the perforation of the drum membrane by fibrin or, in subacute or chronic otitis, by temporary occlusion of the antrum by a polyp. In sinus thrombosis, however, this periodicity frequently appears without tangible reason. It is significant when, for example, acute otitis is associated with acute tonsillitis, and septicemia may be due to the otitis or the tonsillitis. Periodic aural discharge without changes in the perforation of the drum membrane favors the diagnosis of otitic septicemia.

In all of these instances the sinus thrombosis is due to surgical mastoiditis and infection of the sinus wall by contiguity. Since in surgical mastoiditis three to four weeks must pass before a simple mastoid operation is indicated, sinus thrombosis in these

⁷Acute otitis indicates an acute inflammation of the tympanic cavity and pneumatic cells of the temporal bone due to a common cold or influenza. In acute otitis due to scarlet fever, measles and similar infections, intracranial complications, including sinus thrombosis, are not common.

cases usually causes the initial symptoms in the third to fourth week of the otitis. In contrast with these are cases in which acute otitis is associated at the onset with daily rises of temperature and eventually with chills. These cases occur particularly in children and young persons and in some epidemics of influenza. Examination confirms the diagnosis of recent otitis. The drum membrane is red and bulging; occasionally vesicles are seen. There is a moderate amount of serosanguineous discharge, with tenderness and edema at the tip of the mastoid, but there is no sagging of the superior and posterior walls of the canal. X-ray examination usually shows a mastoid which is not well pneumatized. The cells are hazy, but there is no evidence of bone destruction. Occasionally bacteremia is found, and the leukocyte count is normal or moderately increased. Cases of this type offer a trying diagnostic problem. The mastoid is involved, but there is no surgical mastoiditis. This involvement of the mastoid has been called mastoidism. It is particularly common in children up to 10 years, is noted in the first week of severe acute otitis and is not necessarily a precursor of surgical mastoiditis. On the contrary, mastoidism usually subsides spontaneously during the second week of the otitis and may or may not be superseded by a surgical mastoiditis in the third or fourth week. Although mastoidism usually causes mastoid symptoms, it does not cause daily elevations of temperature and chills. These symptoms, when present, are due either to an infection of the lateral sinus or to an infection which does not originate in the ear. For example, in one case of recent acute otitis the spiking temperature was caused by periarthritis nodosa.

Malaria and typhoid fever formerly hampered differential diagnosis. The differentiation of incipient sinus thrombosis and incipient erysipelas is still difficult because in erysipelas fever and chills may precede the erythema. In incipient erysipelas, however, the skin over and near the mastoid is often tender and hypersensitive, whereas in incipient sinus thrombosis there is tenderness only at the tip of the mastoid. Furthermore, in incipient erysipelas the lymph nodes on and near the mastoid are often

involved, whereas in sinus thrombosis they are not inflamed in the incipient phase. If thrombophlebitis is advanced and the thrombosis extends toward the jugular vein, the cervical nodes may become swollen and tender and contain bacteria. Frequently they form a palpable cord which should not be mistaken for a thrombosed jugular vein. If all of these findings do not allow a differential diagnosis, in one to two days erythema will appear if erysipelas is causing the fever. Differential diagnosis of septic sinus thrombosis and bacterial endocarditis may be difficult in rare instances.

Although fever and chills in recent acute otitis may be caused by infections originating elsewhere in the body, time should not be wasted seeking these other sources. Many otologists argue vehemently against the concept of sinus thrombosis caused by recent acute otitis plus mastoidism. Also, neither the surgical findings nor the postoperative course shed light on the pathologic process in these cases. In fact, surgery reveals almost uniform changes: marked hemorrhage from the mastoid, a moderate amount of pus, a normal or almost normal sinus wall and, occasionally, a small perisinus abscess. Convalescence also is almost uniform: immediately after the operation further elevation of temperature, then fall by lysis and, finally, recovery. Obviously these observations imply that surgery had no definite influence on the course of the infection.

A critical review of these cases leads to the conclusion that the infection probably spreads by continuity along anastomosing blood vessels of the mastoid toward sinus and jugular bulb, as in osteophlebitis pyemia. Whether or not the inflammation actually extends to the sinus or jugular bulb is apparently of minor importance; in any case, the septicemia is due to a soft tissue infection without distinct absorption of bone. If this concept is correct, a simple mastoid operation can hardly arrest the spreading infection since the blood vessels, believed to serve as pathways, extend over a large area of the temporal bone. Theoretically, chemotherapy should be superior to surgery in these instances. This theory will probably be tested in the next epidemic.

In mastoiditis and sinus thrombosis caused by pneumococcus type III, the sinus thrombosis seldom causes distinct systemic symptoms. The temperature may be normal (p. 160), and the diagnosis of sinus thrombosis can rarely be made, but the diagnosis of surgical mastoiditis should not be missed (p. 163). Mastoid operation usually discloses extension of the infection to the sinus, commonly by contiguity.

CHRONIC OTITIS.—Chronic otitis is usually classified as (1) a chronic inflammation of the mucosa alone, with central perforation of the drum membrane and odorless secretion, and (2) a chronic inflammation involving both mucosa and underlying bone and presenting a marginal perforation, usually a foul discharge and frequently the formation of cholesteatoma. The concept that chronic inflammation of the bone exclusively causes intracranial complications, including sinus thrombosis, whereas mucosal inflammation is harmless is not wholly correct. Exceptionally, chronic mucosal infection causes intracranial complications, particularly if the chronic otitis has produced adhesions and an acute infection traveling through the eustachian tube or the drum perforation settles between the adhesions. In that event pus between adhesions may lead to intracranial complications. Chronic otitis with bone involvement is more likely to cause intracranial complications in general, and sinus thrombosis in particular, especially when associated with cholesteatoma of the tympanic cavity. Such otitis is never stationary as long as there is any discharge; it advances rather slowly, but continually, toward the dura. The progress is accelerated (1) if there is retention of pus and (2) if there is an acute exacerbation.

Occasionally pus is retained in the epitympanum and antrum with cholesteatoma. The infection may destroy the bone and extend toward the sinus, causing a sudden rise of temperature and eventually a chill. This occurs even though the otoscopic findings in the tympanic cavity are unchanged and there are no symptoms of an acute exacerbation. In fact, otitic sinus thrombosis is probably the only intracranial complication which is due not infrequently to simple retention of pus without additional

acute exacerbation. Usually, however, in otitic intracranial complications the acute exacerbation is more important than simple retention of pus. The acute exacerbation indicates that in chronic otitis caused by a mixture of bacteria, the virulence of one strain, most frequently streptococci, is increased by a superimposed infection which invades the tympanic cavity. There are considerable increase of discharge, expelled from the tympanic cavity with pulsation, pain in the ear, headache, decrease of hearing, tinnitus and vertigo. The mucosa of the tympanic cavity is red and swollen. With association of acute exacerbation and systemic symptoms, the diagnosis of sinus thrombosis is almost certain. The importance of this association is exemplified in the following case.

A young woman had a bilateral ethmoidectomy performed by another rhinologist. The operation on one side was followed by uneventful recovery, but on the other side septicemia developed, metastasizing to the knee. The surgeon considered the septicemia to be of rhinogenous origin. When I saw the patient for the first time the cavity in the ethmoid area was smooth and well epithelialized. The middle turbinates had been removed; the dura was not exposed, and there was no pus. Further examination revealed an acute exacerbation of chronic otitis on the left side which had not been noted by either the surgeon or the patient. A radical mastoid operation and removal of a thrombus from the sinus led to recovery.

To sum up, an acute exacerbation of chronic otitis is important in the diagnosis of intracranial complications of otitic origin. One should not be too dogmatic, however, particularly in cases of sinus thrombosis. Not every instance of acute exacerbation indicates sinus thrombosis, and not every case of sinus thrombosis is associated with an acute exacerbation. Not infrequently an acute exacerbation has subsided by the time the patient notices a rise of temperature and eventually a chilly sensation. Thus, the patient may report that his ear condition had apparently improved when he first noted fever and chills.

AFTER SURGERY ON THE TEMPORAL BONE.—Traumatic sinus thrombosis caused by mastoid surgery is discussed on page 151. The following discussion is limited to sinus thrombosis which appears after mastoid surgery without being caused by it.

Sinus thrombosis may develop immediately or a long time, occasionally several years, after mastoid surgery. The first type is not uncommon; the second is rare. The first type might be called "protracted type of sinus thrombosis."³ The clinical course in many respects resembles that of osteophlebitis pyemia (p. 148). Before the operation both types of infection may cause symptoms of an intracranial complication, such as fever, discharge of irregular intensity and headache. Nevertheless, the symptoms are not so marked as to allow a definite diagnosis of sinus thrombosis. At operation, the exposed lateral sinus appears normal or almost normal in either infection, but after the mastoid operation there is a definite divergence of the clinical course. Although in osteophlebitis pyemia the symptoms of septicemia rapidly subside following simple exposure of the lateral sinus, in protracted sinus thrombosis the contrary occurs. One or several days after the simple mastoid operation the temperature rises sharply (alarining fever; *Komplikationszacke*). In traumatic sinus thrombosis this type of fever occurs one to two weeks after the mastoid operation (p. 152). Chills may or may not be associated with the fever. When, two to four days after the mastoid operation, the lateral sinus is re-exposed the wall is covered by granulations resembling those usually found in a mastoid cavity at this stage. For this reason, one is surprised to find a thrombus or frank pus in the sinus which was apparently normal a few days before. With proper drainage of the pus and thrombi, the outlook for protracted sinus thrombosis is as good as that for osteophlebitis pyemia. Microscopic examinations of temporal bones in such cases are not available because most patients recover so that the pathogenesis of this type of sinus thrombosis is not exactly determined. However, it can be reasonably inferred that there was thrombophlebitis prior to the mastoid operation, perhaps in the jugular bulb or at a site not exposed at operation, and that the simple mastoid operation had a provocative role.

The rare occurrence of intracranial complications, including

³The term latent sinus thrombosis cannot be applied here, for this term is usually applied in instances of sinus thrombosis with an afebrile course.

sinus thrombosis, several years after a radical mastoid operation may be related to a recurrence of cholesteatoma or acute infection of the tympanic cavity. Intracranial complications are more common after simple than after radical mastoid operations for obvious reasons. The radical operation is a standardized procedure. If a technical error is made, the result appears immediately or soon after the operation. The simple mastoid operation must be adjusted to the structure of the mastoid process in the individual case. With marked pneumatization, thorough evacuation of all cells is impossible. Even with average pneumatization, postoperative x-ray films reveal a number of cells remaining despite the surgeon's attempts at thorough exenteration. The remaining cells may become reinfected even several years after the operation and eventually serve as the source of an intracranial complication. Sinus thrombosis may also follow minor manipulations in the tympanic cavity such as extraction of polyps and even probing or irrigation of the cavity. Obviously these manipulations do not actually cause sinus thrombosis; rather, they cause a sinus thrombosis to become manifest. Therefore manipulations in the tympanic cavity should be avoided if a radical mastoid operation is indicated.

Attempts to visualize sinus thrombosis on the x-ray film by injecting a radiopaque medium into the sinus have not progressed sufficiently to make the procedure of practical value.

PROGNOSIS

Broadly speaking, thromboses of dural sinuses tend to form connective tissue and to undergo spontaneous cure. This fact is, however, only of academic interest, since the formation of connective tissue is slow and seldom prevents a fatality from septicemia. Consequently, in the past, prognosis was concerned only with cases in which an operation was performed. The mortality rate was between 30 and 40 per cent, although the actual figures were probably somewhat lower, since the statistics included many cases in which operation was performed in an advanced stage. Chemotherapy now gives patients more than the limited choice

between spontaneous cure and operation, since in certain types of sinus infection chemotherapy is successful. In sinus thrombosis, whether chemotherapy should be instituted first or postponed until the postoperative period depends primarily on the experience and judgment of the surgeon and his knowledge of the principal factors influencing the prognosis for sinus infection: (1) type of infection; (2) diseases of heart and blood vessels and (3) meninges and brain, (4) metastases; (5) time of operation.

The concept that sinus thrombosis in acute otitis has a better prognosis than sinus thrombosis in chronic otitis is inadequate, because there are different types of sinus thrombosis in both acute and chronic otitis and for each type the prognosis is different. Septicemia in recent acute otitis, which is due usually to osteophlebitis pyemia (p. 148) and not to actual sinus thrombosis, has, in general, a favorable outlook. There is often spontaneous cure. Nevertheless, formerly a mastoid operation was usually performed. At present, chemotherapy must be given a trial before operation. Many such cases will probably be cured by chemotherapy alone. In contrast, with septicemia that develops three or four weeks after acute otitis, operation should be performed promptly and chemotherapy given afterward. Prognosis must be guarded, even though it is more favorable than for sinus thrombosis due to an acute exacerbation of chronic otitis, particularly with cholesteatoma. In protracted sinus thrombosis (p. 192) the outlook is, as a rule, good if reoperation is performed at the proper time and chemotherapy is instituted subsequently.

The less favorable prognosis for chronic otitis, particularly with cholesteatoma, apparently is due to the frequent presence of anaerobic bacteria or bacteria which ordinarily act only as saprophytes, such as *B. coli* and *B. proteus*. If these bacteria cause an intracranial complication during an acute exacerbation of chronic otitis, the outlook is impaired considerably. Of all types of sinus thrombosis, the traumatic type (p. 151) has the least favorable prognosis, even when operation is performed at the proper time. Chemotherapy will probably be more efficacious in preventing than in curing traumatic sinus thrombosis.

The condition of the heart and blood vessels has considerable influence on the prognosis for sinus thrombosis. It is often not properly evaluated, although it is primarily responsible for the more favorable prognosis in young people, with a healthy heart muscle and normal blood vessels, than in aged patients. Another factor, related closely to the condition of the heart and blood vessels, is the body temperature following surgery on the mastoid process and sinus. If, in a patient with high fever due to otitic septicemia, the operation is properly performed, the temperature gradually decreases by lysis provided heart and blood vessels are normal. If, however, immediately after surgery the temperature falls abruptly to normal or even becomes subnormal, heart failure or vasomotor paralysis is likely to be the cause, and the prognosis is less favorable.

Purulent meningitis, encephalitis and brain abscess associated with sinus thrombosis render the prognosis extremely unfavorable except in cases of serous meningitis or external and internal pachymeningitis of the posterior cranial fossa.

Metastases localized in the meninges, brain or abdomen impair the prognosis. This is not necessarily the case if they are localized in the joints or muscles, although ankylosis of the joint may result. A single metastasis to the lung is not necessarily unfavorable if proper treatment of the lungs is instituted.

If the diagnosis of sinus thrombosis is established and it is evident that chemotherapy is useless, surgery should not be postponed in any circumstances.

TREATMENT

Conservative treatment is discussed on page 163. Surgery includes operation on the mastoid, sinus and jugular vein.

The mastoid operation does not fall in the scope of this discussion. However, it should be emphasized that the operation on the sinus should be performed only after the mastoid operation is completed, so as to avoid injury and/or infection of the sinus during the mastoid operation. Occasionally, before draining the mastoid antrum, surgeons create a funnel-like defect in the mas-

toid process and expose the sinus at the tip of the funnel (Fig. 45). This never permits proper inspection of the sinus, and since the gouge is driven into the bone perpendicularly to the sinus, injuries to the sinus readily occur. When this happens it is extremely difficult to put a tampon on the injured sinus through the funnel-like bone defect.

I know of a fatal case of this type resulting from loss of blood through the mastoid antrum and eustachian tube into the pharynx.

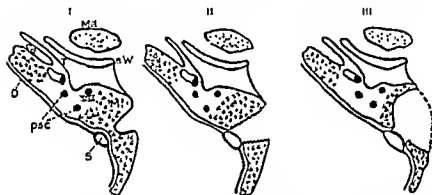


FIG. 45.—Procedures for exposure of lateral sinus: *I* and *II*, incorrect methods. *III*, correct method. *D*, dura of posterior cranial fossa, *psc*, posterior semicircular canal, *S*, lateral sinus; *M*, mastoid process, *VII*, facial nerve, *aW*, anterior wall of external auditory canal, *Mo*, mandible, *T*, tube, *ca*, carotid canal (Adapted from Alexander.)

Several futile attempts were made to arrest the bleeding by putting tampons on the sinus. No attempt was made to expose the sinus by the proper technic and then to place a tampon on the bleeding sinus.

The sinus operation consists of exposure of the bony plate covering the sinus. This should be done regardless of whether there is a defect of the bony plate or not. When the bony plate is prepared it must be removed. The gouge is driven into the bone tangentially to the sinus until a defect is created the size of a pea (Fig. 46). This first exposure should always be made between the knees of the sinus, where the sinus wall is comparatively thick. In no circumstance should it be created at the inferior knee, where the wall is thin. To enlarge the defect, a small rongeur is used. If exposure of the sinus is complete, there should be no

empty space between the sinus and the margins of the bone defect. An empty space is usually due to a small bone chip which slipped between the bone and the sinus wall and which must be removed by enlarging the defect to avoid hemorrhage or sinus thrombosis after operation.

If the bony plate was absorbed by infection prior to the operation, the defect should be enlarged. Exposure of the sinus wall

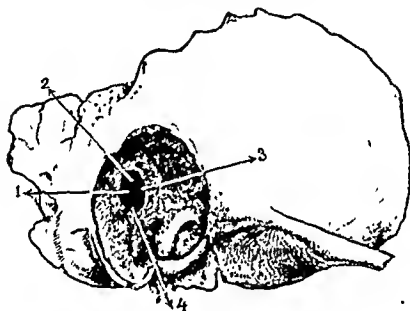


FIG. 46—Directions of exposure of lateral sinus (After Alexander.)

in all circumstances should be continued until normal wall is reached, as indicated by a dark blue or grayish blue color, depending on the thickness of the wall. This must be accomplished in the direction of the torcular unless the patient's condition does not permit completion of the operation. Toward the jugular bulb it is not always possible to reach normal wall unless a bulb operation is performed. This, however, should be done only with strict indications (p. 214). Frequently it suffices to expose the sinus to its junction with the bulb. After the sinus is exposed, whether or not it should be further explored depends primarily on the clinical findings and secondarily on the surgical findings. It is definitely

indicated if pus escapes through a fistula of the sinus wall, if there is necrosis of the wall or if the sinus is collapsed. Otherwise the clinical findings are decisive. For example, cholesteatoma and definite septicemia indicate exploration even though the wall is smooth. Exploration may be postponed, even though the wall is covered by a layer of granulations, when there is no clinical evidence of septicemia. When in doubt, I prefer exploration.

The sinus can be explored by palpation, puncture or incision. Palpation of the sinus with the finger is not as reliable as the other methods. For puncture, a short bevel needle is introduced at an acute angle to the sinus where the thrombus is thought to be located. To avoid injury to the cerebral wall the needle should never be introduced perpendicularly to the sinus. Puncture permits the definite diagnosis of thrombus if no fluid or if pus is aspirated. If blood is aspirated the needle may have perforated a mural thrombus. Occasionally a mural thrombus can be more definitely diagnosed by the following reasoning. If a normal sinus is punctured and the needle withdrawn, blood escapes through the perforation. If the needle has penetrated a mural thrombus, it obliterates the perforation when the needle is withdrawn and no blood escapes through the perforation although blood was aspirated. This observation allows the tentative diagnosis of mural thrombus, and the sinus should be incised. Since, after withdrawal of the needle, blood may escape through the perforation because the mural thrombus is at another site in the sinus, puncture should be repeated at various sites of the sinus wall if clinical symptoms suggest sinus thrombosis.

The incision is made along the longitudinal axis of the lateral sinus in the center of the wall. It should never be made near the margins of the bony defect (Fig. 47). If the incision in the center does not disclose a thrombus, the incision (and also the puncture) closes rapidly owing to the formation of a small thrombus which soon becomes organized. An infection of the sinus is rare. If the incision is made near the margin of the bony defect and no thrombus is discovered and the sinus wall is thin, part of the incised sinus wall may slip beneath the bone while the other part

does not move, and the gap remains patent for a long period. The result is obstinate hemorrhage from the sinus which is arrested only with great difficulty. With other otologists, I believe that obstinate hemorrhage from an incised or punctured sinus is

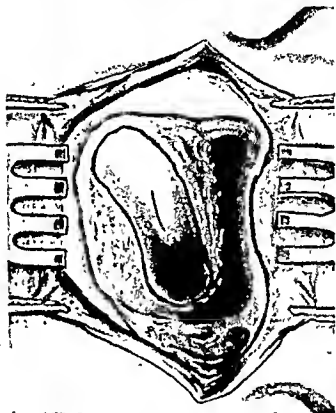


FIG. 47—Exposure of lateral sinus. Solid line indicates correct incision, dotted line indicates incorrect incision

due more often to errors of surgical technic than to changes in blood chemistry. The incision should be gradually deepened down to the endothelium. The emptying of the sinus by compression with tampons before the incision is made is usually superfluous except in some cases of thrombosis of the jugular bulb. Incision yields practically the same results as puncture. It does not permit exclusion of a mural thrombus when blood escapes from the incision. Occasionally the following observation indicates a mural thrombus: if a normal sinus is incised, blood immediately spurts

out when the endothelium is incised, but if the sinus wall is gradually incised down to a mural thrombus, only drops of blood escape at first and then, after a few seconds, blood spurts out as from a normal sinus. In these instances the mural thrombus is probably expelled by pressure from within.

The choice of incision or puncture is a matter of personal preference. I prefer incision for various reasons. Incision permits better judgment concerning the thickness of the sinus wall. Occasionally the wall is so thickened that the sinus lumen is markedly narrowed, in which case puncture is not advisable. The same is true when the sinus is collapsed, the wall is gangrenous or there is a fistula of the wall which must be enlarged by incision. Furthermore, puncture seems to be hazardous when there is marked external pachymeningitis of the posterior cranial fossa as well as marked sinus phlebitis. In cases of this type it might be difficult to differentiate dura from sinus and a puncture might easily penetrate the subarachnoid space and even the brain. A careful incision diminishes, although it does not eliminate, the risks of the procedure.

If exploration discloses a normal sinus or a mural thrombus, a tampon is placed on the opening of the sinus wall. Great pressure is not necessary, nor is it advisable to insert the tampon into the lumen. Chemotherapy is administered and the tampon is removed two days after operation. Hemorrhage is rare provided the incision was correctly performed. Some surgeons suggest leaving the tampon in place for a longer period to encourage the formation of an obliterating thrombus when incision disclosed a mural thrombus. It is argued that an obliterating thrombus can be removed more easily than a mural thrombus, but I consider this procedure hazardous. If an obliterating thrombus is discovered, the incision of the sinus wall must be enlarged as far as the wall is involved. This coincides approximately with the length of the thrombus. Then the thrombosis is gently removed with a curet. Occasionally the ends of the thrombus can be removed by introducing a suction tube into the sinus. I have never ligated the sinus at the torcular, as suggested by several surgeons. The

operation is perfectly performed if, after removal of the thrombus, blood spurts from both the central and the peripheral part of the sinus. Some surgeons prefer partial thrombectomy and do not remove the ends of the thrombus when they appear to be firm and solid, contending that the ends are sterile and prevent bacterial invasion of the blood stream. In general, I do not favor this procedure. The gross appearance of a thrombus does not permit conclusions concerning its bacterial content (p. 154), and unless the thrombus has disintegrated into pus, only a bacteriologic examination can determine its content. However, there is no time for this type of examination during the operation. Furthermore, the ends of the thrombus will hardly remain sterile after the sinus is opened and are brought in contact with the infection in the mastoid.

Although partial removal of a thrombus from the sinus is not a sound surgical principle, total removal is not feasible in some instances and is not necessary in others. It is not necessary when there are no signs of septicemia. It is not necessary or feasible when the thrombus is replaced by granulations which are adherent to the wall of the sinus and could be removed only by forceful curettage, which is never permissible.

Often the hemorrhage from the peripheral part of the sinus is satisfactory but there is no bleeding or insufficient bleeding from the jugular end. Owing to the curvature of the inferior knee and jugular bulb the thrombus cannot be removed from the bulb either by introduction of a curet or by aspiration. Occasionally the following maneuver is effective: when the sinus is incised, *pressure on the internal jugular vein causes a sudden rise of venous pressure which may expel a thrombus from the inferior knee.* Failure of this maneuver may necessitate an operation on the jugular bulb for removal of the thrombus, but first a trial should be given to less radical procedures, particularly ligation of the internal jugular vein.

After the operation a tampon is placed on the sinus, not into it. Over the tampon the margins of the skin incision are approximated by one or two sutures, and moderate pressure is exerted on

the sinus. Chemotherapy is instituted. Two days after the operation an attempt should be made to remove the tampon under aseptic conditions. Another sterile tampon must be prepared, to be placed on the sinus if hemorrhage occurs. In this event another attempt should be made to remove the tampon the next day, when the procedure usually succeeds.

It must be frankly admitted that this surgical procedure cannot be considered radical. Even if the entire thrombus were removed, and even if a part of the sinus wall were excised, total removal of the inflamed sinus wall could not be accomplished. The inflammation always extends farther than the gross appearance indicates. An attempt to remove a great part of the sinus wall might result in an opening of the subdural spaces. For this reason, the inflammation of the sinus wall may spread despite surgery, causing the formation of more thrombi. Consequently, in every case of sinus thrombosis chemotherapy must be instituted, no matter how radical the operation is considered by the surgeon. Several days after fever has subsided and chemotherapy has been discontinued, there may be another rise of temperature for one to two days without further symptoms of septicæmia, followed by final descent to normal. Whether this rise of temperature is caused by the sulfonamide or by the inflammation cannot be stated. In any event, fever several days after a sinus operation does not indicate a spreading inflammation provided it continues only one to two days and there is no infection of the skin incision. With recovery the thrombus is replaced by connective tissue. Occasionally the emissaria and other veins of the bone marrow become dilated to facilitate the outflow of venous blood from the skull.

The third surgical procedure is ligation of the jugular vein. An incision about 6 cm. long is made along the anterior border of the sternocleidomastoid muscle in the middle third of the neck. Here the vein is nearest the surface and is discovered immediately beneath the muscle. The vein is prepared for ligation, and two silk ligatures are placed 2-3 cm. apart. To avoid eventual spread of thrombophlebitis along the jugular vein, the vein is severed

between the ligatures. The severing should be done gradually so that the central end does not slip beyond the central ligature; otherwise the vein retracts rapidly, hemorrhage is profuse and there is danger of air embolism. If an air embolism occurs the central portion of the vein must be compressed and the patient's head bent forward. After ligation, the silk thread at the central part of the vein is cut short; the thread at the peripheral part is left long so that it can be secured if the postoperative condition requires the opening of the peripheral portion of the vein. Ligation should be performed, if at all possible, above the common facial vein, so that it does not interfere with the outflow of blood from the facial veins. Some surgeons place one ligature above and another below the facial vein and shut off the facial vein with a third ligature. I prefer the first method. Glands adjacent to the vein should be left in place if normal, but should be removed if enlarged and inflamed. If the vein presents phlebitis or thrombosis, ligation must be done below the inflamed area. In thrombosis of the jugular vein which extends beyond the clavicle, the vein is incised down to the clavicle and the wound left open. Ligation is not performed, since this would require an operation on the clavicle, which should be avoided.

If after the operation the temperature falls by lysis, the peripheral ligature is cut short in three or four days and the neck incision allowed to close. If septicemia persists, the peripheral ligature may be removed two days after the operation, at the earliest, and the peripheral part of the vein sutured to the skin. This is the jugular-skin fistula. Usually there is no bleeding from the vein if there are no anatomic abnormalities, since the jugular bulb does not receive blood either from the lateral sinus, which was incised at the mastoid operation, or from the inferior petrosal sinus, which is probably obliterated by a thrombus. In some cases two days is too short an interval and hemorrhage occurs when the peripheral ligature is removed. The ligature must then be renewed and the next day a second attempt should be made to open the vein. Bleeding from the jugular vein five or six days after the operation suggests that, in addition to the common facial vein,

branches from the cranial portion of the jugular pour blood into the vein.

Ligation of the jugular vein is seldom difficult under typical conditions. If the vein is thrombosed or, to a certain extent,

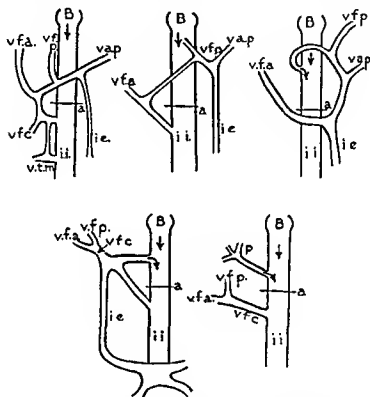


FIG. 48.—Anomalies of internal jugular vein (*B*, jugular bulb, *ie*, external jugular vein, *Vap*, posterior auricular vein, *Vfp*, posterior facial vein, *Vfa*, anterior facial vein, *Vfc*, common facial vein, *Vtm*, mesial thyroid vein, *P*, pharyngeal vein; *a*, ligature. Arrows indicate the flow of blood.

necrotic it is difficult to recognize the vein and it should be prepared near the clavicle, where it is most likely to be normal. Anatomic abnormalities also increase the difficulty (Fig. 48), but they are not common. Occasionally there is a duplication of the internal jugular vein, but this usually involves the cranial portion and rarely extends to the site of the ligation.

Ligation of the internal jugular vein carries certain hazards.

The possibility of an air embolism was previously mentioned. Another risk is permanent recurrent laryngeal paralysis. The opponents of ligation stress the possibility of causing spreading thrombophlebitis or passive hyperemia of the brain.

The changes which take place when the jugular vein is severed between two ligatures are not definitely known. One can reasonably assume that changes in the blood circulation and eventually the formation of a blood clot will occur, at least in the peripheral portion of the vein. Some surgeons argue that the changes in circulation may cause infected particles to be disengaged from a thrombus in the jugular bulb and to be carried into the body. Furthermore, the clot which after ligation may form in the peripheral portion of the vein is believed occasionally to become infected and to extend along the inferior petrosal sinus into the cavernous sinus. In my opinion these arguments are somewhat theoretical. Cases which might be considered as proving this concept are rare and do not permit a generalization. Furthermore, ligation of the jugular vein is not necessarily the final step of surgery. The peripheral portion of the vein may be opened as early as 48 hours after ligation, which means that untoward effects of the ligation can soon be counteracted. Symptoms of passive hyperemia of the brain occasionally occur if the jugular vein on the opposite side is narrow. Papilledema, retinal hemorrhage and abducens paralysis have been observed under these circumstances. They are particularly apt to appear after jugular ligation in infants or children. A few cases of death subsequent to ligation have been reported. However, in some it is not certain that death was actually due to the ligation, and in others the ligation was performed during a goiter operation and not for a sinus thrombosis. Therefore no definite conclusions can be drawn from these reports. Symptoms of passive hyperemia of the brain are rare, and if they occur they usually disappear without causing permanent ill effects. In obliterating sinus thrombosis, jugular ligation can hardly cause passive hyperemia because the circulation of blood is blocked by the thrombus, regardless of ligation. Even ligation of the vein on both sides

does not necessarily cause permanent symptoms if between the ligation on either side a period of, say, one to three weeks is allowed to elapse or if both sinuses are thrombosed.

To sum up, the risk of ligation of the jugular vein is not great. For this reason, the question is not whether the ligation is hazardous but whether it is necessary. In this respect there is a wide diversity of opinion. Some surgeons have abandoned ligation, whereas others perform it in every case of otitic septiceimia, even doing it before the mastoid operation or without subsequent incision of the lateral sinus. Formerly I followed the latter practice. However, more experience and theoretical considerations have led to a change of view. When the jugular vein is severed, only one of many channels is shut off, even though it is the principal and the shortest channel between sinus and systemic circulation. Other channels may still carry infective material into the body, including the anterior and posterior condyloid veins, particularly the former, which establishes an anastomosis between the jugular bulb and the prevertebral plexus along the deep cervical veins. From the prevertebral plexus the organisms may be carried into the vertebral vein, then into the subclavian vein and ultimately into the heart. Another channel is the inferior petrosal sinus, which may carry the infection into the cavernous sinus by retrograde extension. A third channel is the marginal sinus, which is a continuation of the occipital sinus and establishes an anastomosis of the lateral sinuses on either side. A fourth channel is the external jugular vein, if it originates in the vein of the mastoid emissaria (p. 18). A fifth channel is a vein which sometimes runs from the uppermost part of the internal jugular vein toward the prevertebral plexus. A sixth channel is the transverse sinus on the involved side, which may carry organisms toward the torcular and to the transverse sinus of the other side by retrograde extension. In view of the many accessory channels, ligation of the internal jugular vein cannot definitely prevent a spread of the infection. But also, the operation on the sinus cannot entirely prevent a spread of the periphlebitis, nor can a simple mastoid operation on a well pneumatized

mastoid definitely prevent a flare-up in an infected cell not reached at operation. Therefore jugular ligation accomplishes no more and no less than many other surgical procedures performed to combat infections of the ear. The results of the sinus operation plus jugular ligation are remarkably good even without chemotherapy. At the Allgemeine Poliklinik in Vienna, 155 patients were treated up to 1928 according to these principles, and the mortality rate was only 21 per cent, as against the average mortality rate of 35 per cent. For this reason, there is apparently no reason to abandon jugular ligation.

Admittedly, some patients can be cured without ligation, particularly if chemotherapy is employed. The following considerations seem to be practical. The decision to ligate the internal jugular vein depends on (1) the patient's general condition, (2) the presence of metastases and (3) the pathologic changes in the jugular vein and lateral sinus. Some patients with septic sinus thrombosis due to cholesteatoma of the tympanic cavity have a rapid downhill course. The surgeon must decide whether the patient will tolerate a sinus-jugular operation or only a sinus operation. If metastases are present, ligation should be performed immediately after, or even before, the sinus operation to prevent the formation of more metastases. Only in young patients with osteophlebitis pyemia and metastases around the joints can ligation be postponed. Girls particularly should be spared, if possible, a conspicuous scar of the neck. In infants, jugular ligation should be avoided as long as possible. In cases of thrombosis of the jugular vein, ligation must precede the sinus operation. *These cases are not common and offer a difficult diagnostic problem, since neither palpation of swollen glands nor palpation of a cordlike structure in the neck allows a definite diagnosis of thrombosis of the jugular vein.* However, a diffuse swelling of the neck, definite tenderness along the jugular vein and complaint of pain on movement of the head justify a tentative diagnosis. In the lateral sinus, the further procedure depends on the findings at the inferior knee and in the jugular bulb. If the wall at the inferior knee seems normal or only slightly changed

and if, after incision of the sinus, blood spurts forcefully from the jugular bulb, ligation can be omitted at the time of the sinus operation. Ligation usually is not necessary if the inferior knee contains granulations or connective tissue, but not pus or thrombi, and if there were no chills or septic fever immediately before operation. In such cases, recovery may occur without jugular ligation, even though a thrombus above the inferior knee has disintegrated into pus and the pus escapes through a fistula of the sinus wall into the mastoid. On the other hand, ligation must be performed if the bony frame of the jugular bulb or the sinus wall at the inferior knee presents marked changes, if pus escapes from the bulb and if it is not possible to remove all thrombi from the jugular bulb and obtain free bleeding from the bulb.

Often the jugular vein must be ligated several days after the sinus operation if the infection advances despite operation, as shown by continuation of spiking temperature. If the fever is suppressed by chemotherapy, progress of the infection may be indicated by delay in convalescence, failure of the retroauricular incision to close, abundant discharge from both the tympanic cavity and the mastoid, cerebral symptoms and metastases. The patient should be permitted out of bed for one day. If he responds to this physical exercise with a rise of temperature, one may assume that the sinus thrombosis (or the intracranial complication, in general) is still active and jugular ligation is indicated, even several days after the sinus operation.

If the vein is inflamed the neck incision is left open. If the vein is normal, the lower angle of the incision is left open and the remainder closed. Through the lower angle iodoform gauze is inserted and the long ligature on the peripheral portion of the vein is fixed to the skin with adhesive.

Rarely, jugular ligation on both sides is indicated. If insufficient time elapses between the first and the second ligation and if the second ligation shuts off a sinus which is not thrombosed, alarming symptoms may occur, such as unconsciousness, headache, vomiting, slow pulse, convulsions, pareses, glycosuria.

venostasis of the orbit and face and papilledema. The following procedures may prevent these symptoms. Assuming that the right jugular vein was previously shut off and that the left jugular vein is to be ligated, the risk is not great if the left lateral sinus is obliterated by a thrombus. If, however, the left sinus contains blood, a tampon should be placed on the left sinus. If this procedure is followed by persistent headache, nausea and drowsiness, ligation of the left vein would be hazardous. X-ray examination of the jugular foramina has been suggested to determine whether there is conspicuous difference in the size of the two jugular bulbs.

THROMBOPHLEBITIS OF THE JUGULAR BULB PATHOLOGY

The jugular bulb may be involved primarily or secondarily. Secondary involvement occurs when thrombophlebitis of the



FIG. 49.—Thrombophlebitis of jugular bulb (*Th*). *M*, tympanic cavity; *G*, granulation tissue; *B*, floor of tympanic cavity.

lateral sinus or jugular vein extends into the bulb by continued growth of the thrombus. Occasionally, secondary involvement is due to particles carried by the blood from a sinus thrombosis into the jugular bulb, where they adhere to the wall. Primary

thrombosis of the jugular bulb occurs when an infection in its vicinity invades the wall. In this respect the hypotympanum and peribulbar cells are important. Infections of these cavities may extend toward the bulb either by contiguity or by continuity and may cause a peribulbar abscess or thrombophlebitis of the bulb, or both (Figs. 49 and 50). A bulb which bulges markedly into



FIG. 50—Thrombus in jugular bulb, partially organized and containing newly formed blood vessels (B). *hPy*, posterior surface of petrous bone, *M*, marrow space, inflamed and perforating the dome of the bulb.

the tympanic cavity is more often involved than a shallow one, which usually is covered by a thick layer of bone.

Thrombosis of the bulb is either mural or obliterating. An obliterating thrombus may extend (1) into the jugular vein, (2) into the lateral sinus, (3) into the condyloid veins or (4) into the inferior petrosal sinus. If it extends into the jugular vein it extends simultaneously into the lateral sinus, and often it is not possible, nor is it necessary, to determine whether the thrombophlebitis originated in the bulb or in the sinus. However, when the sinus plate is normal and the wall presents inconspicuous changes but the lumen is filled by a thrombus, one may assume tentatively that the thrombophlebitis originated in the bulb. If the thrombophlebitis extends into the condyloid veins, a deep abscess of the neck and even erosion of the atlanto-

occipital joint may ensue, with involvement of the hypoglossal nerve. If the thrombophlebitis extends into the inferior petrosal sinus, the abducens nerve is involved and eventually the infection spreads into the cavernous sinus.

SYMPTOMATOLOGY

In the secondary type of bulb thrombosis the symptomatology is essentially that of lateral sinus thrombosis and is the same when the primary type extends into the lateral sinus or jugular vein. In the initial phase the diagnosis of primary bulb thrombosis is almost invariably tentative. The systemic and general brain symptoms do not differ from those of sinus thrombosis. Among the focal brain symptoms are hoarseness, bradycardia, difficulty in swallowing without pathologic change in the throat and spasm of the sternocleidomastoid and trapezius muscles. These symptoms occur if the glossopharyngeal, vagus and accessory nerves, which run through the foramen jugulare, are involved either by inflammation or by compression. These symptoms are not common, except the difficulty in swallowing, which may be due to a retropharyngeal abscess originating in the base of the petrous bone. Primary bulb thrombosis is more frequently due to acute than to chronic otitis. Among six examples of bulb thrombosis in my clinicoscopic collection, three were of the primary type and three of the secondary type. The three cases of primary thrombosis were due to acute otitis and the three of secondary type to chronic otitis. This observation is somewhat surprising since cholesteatoma is frequently localized in the hypotympanum and theoretically should more often cause an infection of the jugular bulb. However, the thickened and sclerotic floor of the tympanic cavity probably prevents infection of the jugular bulb in these instances. The acute otitis which causes bulb thrombosis does not differ from common acute otitis except that it is associated with fever. However, as in lateral sinus thrombosis, the course in bulb thrombosis may be afebrile, and even more frequently than in the former.

The symptoms are few, but sufficient to offer indications for

operation on the mastoid. Even during the operation definite diagnosis is difficult except in certain cases of obturating thrombosis of the jugular bulb. In these exceptions two observations are significant. One is the discovery of an empty jugular vein. This is rare, since it can occur only with occlusion of both the jugular bulb and its tributaries, particularly the inferior petrosal sinus. The second, more common, observation is made when the lateral sinus is incised close to the inferior knee and the peripheral part of the sinus is shut off by a tampon; the diagnosis of bulb thrombosis can be made if pus pours from the bulb or if neither blood nor pus appears.

Although diagnosis of obturating thrombosis of the jugular bulb can be made eventually, diagnosis of mural thrombus is impossible without exposure of the bulb. For this reason the diagnosis is commonly made by exclusion: one may conclude that there must be a thrombus of the bulb when otitis media is associated with septicemia and there is no pathologic change in the lateral sinus. This conclusion is usually, but not always, correct because septicemia may be due to osteophlebitis pyemia or thrombophlebitis of the carotid plexus instead of a mural thrombus.

PROGNOSIS

The prognosis for bulb thrombosis does not differ materially from that for sinus thrombosis. However, thrombophlebitis of the jugular bulb apparently undergoes spontaneous cure more frequently than that of the lateral sinus, as demonstrated in microscopic studies of temporal bones.

TREATMENT

The first question which arises is whether or not there is surgical mastoiditis. If there is no surgical mastoiditis or metastases and the patient's general condition is good despite septicemia, chemotherapy may be tried for a few days. If chemotherapy fails to achieve definite improvement or if surgical mastoiditis develops, the mastoid operation should be performed and the sinus explored. With evidence of bulb thrombosis, a simple mastoid

operation and postoperative chemotherapy will probably suffice, particularly in children, if the patient is in good condition, if there are no metastases, if the septicemia is of short duration and if there is no gross destruction in the area of the jugular bulb. Otherwise the jugular bulb should be surgically drained either through the jugular vein or by operation on the bulb. The first procedure consists of ligation of the internal jugular vein. Some surgeons also advocate irrigation of the sinus-jugular tube through the jugular vein or through the lateral sinus if the peripheral portion of the lateral sinus is closed by granulations or connective tissue and if the sinus does not contain blood. In one case I irrigated with Dakin's solution, but the fluid which entered the bulb from the incised sinus failed to escape through the jugular vein. This was an embarrassing experience, and although the patient ultimately recovered, I never used irrigation again. Some surgeons consider irrigation hazardous because it may carry particles of the thrombus into the tributaries of the bulb and the systemic circulation. For this reason it seems preferable to create a skin-jugular fistula by the technique of Alexander (p. 203) and to keep it open as long as necessary by inserting iodoform gauze wicks in the lumen of the vein.

Different techniques have been suggested for exposing the jugular bulb. The method of Voss is apparently the most practical procedure. After the entire tip of the mastoid process is removed and the lateral sinus exposed, bone removal is continued down to the lowest point of the sinus, where the lateral sinus turns from its perpendicular into its horizontal course to form the inferior knee (Fig. 51). In the area of the inferior knee only small pieces of bone should be removed in order to avoid injury to the facial nerve. At the lower boundary of the inferior knee, the bone immediately above, which is the posterior wall of the jugular fossa, is removed. At this stage the gonge is driven from below upward, and the exposure is extended not more than 0.5 cm. from the lower boundary of the sinus in order to avoid injury to the posterior semicircular canal. At this stage injury to the facial nerve is unlikely because the chiseling is done parallel to

the descending portion of the nerve and mesial to it. The entire posterior wall can be removed up to the roof of the jugular dome. Now the posterior wall of the jugular bulb is incised and partially excised, and the thrombi are removed. This operation should be performed if the posterior wall of the jugular fossa presents

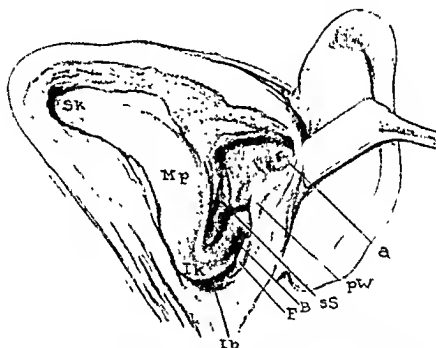


FIG. 51—Exposure of jugular bulb by technique of Voss. *a*, mastoid antrum, *pw*, posterior wall of external auditory canal, *ss*, area of posterior semicircular canal, *B*, jugular bulb, *F*, area of facial nerve, *ib*, inferior boundary of lateral sinus, *ik*, inferior knee of lateral sinus, *mp*, middle portion of lateral sinus, *sk*, superior knee of lateral sinus. (Adapted from Hayman.)

osteitis or if the bulb thrombosis has extended into the condyloid veins, causing an abscess of the neck. Otherwise, ligation of the jugular vein is sufficient for drainage of the bulb.

THROMBOPHLEBITIS OF SUPERIOR PETROSAL AND PETROSQUAMOUS SINUSES

Thrombophlebitis of the superior petrosal sinus is often associated with meningitis and brain abscess. Obviously diagnosis

cannot be made and the thrombophlebitis is found only in microscopic studies of the temporal bone.

Thrombophlebitis of the superior petrosal sinus as a morbid entity is rare, but it occurs occasionally with chronic otitis, particularly with cholesteatoma of the tympanic cavity. Infection extending toward the dura on both sides of the superior angle of the petrous bone causes external pachymeningitis of both middle and posterior cranial fossae which, in turn, may cause thrombophlebitis of the superior petrosal sinus.

Preoperative diagnosis of thrombophlebitis of the superior petrosal sinus is not possible. However, the chills and spiking temperature eventually associated with this type of infection may lead to incorrectly planned surgery. In the presence of these symptoms the otologist is accustomed to think of a complication in the posterior cranial fossa and to expose the lateral sinus. In such instances the lateral sinus is usually normal, and the fever actually is due to infection of the superior petrosal sinus, which runs through the middle, not the posterior, fossa.

The prognosis is good, since thrombophlebitis of the superior petrosal sinus does not cause metastases. Nor does it cause leptomeningitis if the pachymeningitis of the middle cranial fossa is drained at the proper time.

Treatment is surgical and consists of exposure of the dura of the middle fossa and removal of the superior petrous angle.

A rarer condition is thrombophlebitis of a persistent sinus petrosquamosus (p. 26). Diagnosis is made at operation. The prognosis is favorable.

In a boy, aged 8 years, with periphlebitis of the lateral sinus and large external pachymeningitis of the posterior fossa due to acute otitis (*Streptococcus haemolyticus*), a petrosquamosus sinus was found at operation. Although the lateral sinus was covered by a thick layer of granulations, the petrosquamosus sinus was normal. He recovered.

THROMBOSIS OF THE CAVERNOUS SINUS

PATHOLOGY

Thrombophlebitis of the dural sinuses, as mentioned earlier, is usually due to an infection by contiguity. Infection of the

cavernous sinus by contiguity occurs only with osteomyelitis of the sphenoid sinus, osteomyelitis or abscess of the petrous apex and, occasionally, osteitis of the lamina papyracea in infections of the posterior ethmoid. Osteomyelitis of the petrous apex is discussed on page 111. Osteomyelitis of the sphenoid sinus involves the posterior wall especially and causes external and internal pachymeningitis of the dura of the sella turcica, purulent or necrotizing inflammation of the hypophysis and thrombophlebitis of the cavernous sinus. There is often simultaneous infection of the leptomeninges. Rarely, cavernous thrombophlebitis is due to contiguous infection originating in necrotizing osteitis of the posterior part of the lamina papyracea following infection of the posterior ethmoid. Among 66 cases of cavernous thrombophlebitis of rhinogenous, otitic and pharyngeal origin, only 10 per cent were due to infection by contiguity. When cavernous thrombophlebitis is due to infection by contiguity the infection may spread to the orbit, causing orbital phlegmon. Since it is secondary to the cavernous thrombophlebitis, it is called secondary orbital phlegmon.

Infection by continuity is more common than that by contiguity. Venous radicles carry the infection from remote areas into the cavernous sinus either by direct extension or, less frequently, by metastasis. Various venous radicles may serve as pathways to the cavernous sinus. The following venous pathways are of principal importance.

Ophthalmic veins.—These venous radicles carry the infection into the cavernous sinus from the skin of the face, the orbit and the paranasal sinuses. Because the ophthalmic veins anastomose freely with the frontal, angular and anterior facial veins (Fig. 4), infections, usually staphylococci, of the lips, eyelids, nasal orifice, teeth or mouth may be carried into the cavernous sinus. Also, infections originating in the tear sac or the orbit (for example, owing to foreign bodies in the orbit) may travel into the cavernous sinus. Such infections, though common, do not fall in the scope of this volume.

Infection of the ophthalmic veins may follow invasion of the

orbit by a purulent infection of the frontal, anterior ethmoid or maxillary sinus. Such cases are not common, my statistics disclosing an incidence of only 1.5 per cent of orbital infections originating in the paranasal sinuses and causing cavernous thrombosis. Simple orbital infections of paranasal origin without infection of the ophthalmic veins are common, particularly in children and

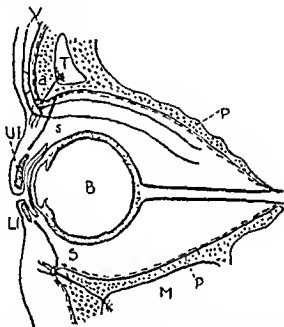


FIG. 52.—Anastomosing veins (a) between mucosa of frontal sinus (T) and upper eyelid (UI) and between maxillary sinus (M) and lower eyelid (LI). B, eyeball; S, orbital septum; P, perosteum of orbit (After Folk and Brunner.)

young people, and are due to infection either by contiguity or by continuity. With infection by contiguity there is osteitis of the anterior part of the lamina papyracea, the floor of the frontal sinus or the roof of the maxillary sinus. Infection by continuity is carried along the anastomosing blood vessels which originate in the mucosa of the various sinuses, run through the bone and ramify in the periosteum of the orbit (Fig. 52). Gross changes in the bone are rare in these cases.

When infection has invaded the orbit there may be collateral hyperemia of the orbit, subperiosteal abscess or phlegmon. Col-

lateral hyperemia is common, particularly with ethmoiditis. It may lead to subperiosteal orbital abscess but usually resolves spontaneously, and simultaneously with the ethmoiditis. It never causes cavernous thrombosis. Subperiosteal abscess indicates an accumulation of pus between the bone and the periorbital (Fig.

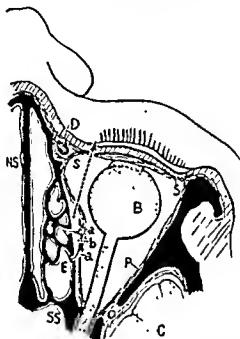


FIG. 53—Horizontal section through nasal septum (Ns), ethmoid (E) and eyeball (B). D, nasolacrimal duct, S, orbital septum continuous with tarsus and periorbital (P), ss, sphenoid sinus, o, superior orbital fissure, c, brain, n, anastomosing veins between ethmoid and periorbital, b, subperiosteal abscess of orbit. Arrow indicates pathway of infection from the abscess in the lids, causing lid edema. (After Folk and Brunner.)

53) and is analogous to an extradural abscess. It is usually associated with collateral hyperemia of the orbit and tends to spread forward toward the orbital septum (Fig 53). Frequently the infection travels through the orbital septum to cause lid edema or abscess. Rarely, if ever, the abscess extends back toward the optic foramen. Usually a subperiosteal abscess must be drained by incision of the orbit. Occasionally it drains spontaneously into a paranasal sinus through a fistula in the bone. Cavernous thrombo-

sis from a subperiosteal abscess of the orbit is unusual. Because the periorbita is highly resistant to infection, the abscess rarely causes necrosis or a fistula of the periosteum; if it does, an orbital phlegmon ensues. The ophthalmic veins are thus exposed to infection, and if they become involved they promptly carry the infection into the cavernous sinus. In such cases phlegmon precedes the cavernous thrombosis and is called primary orbital phlegmon.

With orbital phlegmon not only the ophthalmic veins but the loose connective tissue of the orbit, the nerve sheaths and the orbital muscles may carry the infection toward the periosteum of the base of the skull, causing osteomyelitis and, ultimately, external and internal pachymeningitis with the cavernous thrombosis. Orbital phlegmon is frequently diagnosed when there is only collateral hyperemia or a subperiosteal abscess of the orbit. This is a grave error because an orbital phlegmon nearly always causes cavernous thrombosis, whereas the others rarely, or never, do. Fortunately, orbital phlegmon, which is comparatively common with infections originating in the skin of the face, is seldom caused by an infection of the paranasal sinuses, whereas collateral hyperemia and subperiosteal abscess of the orbit are not unusual sequelae.

Pterygoid plexus.—Recognition of an infection of the pterygoid veins (Fig. 10, p. 23) presents certain difficulties. The pterygoid plexus may become involved from various sources, the principal ones being the tonsils, pharyngeal mucosa, maxillary sinus and teeth. Infection originating in the teeth does not come within the scope of this volume.

Like the ophthalmic veins, the pterygoid veins run through a space, the pharyngomaxillary, filled with loose connective tissue and nerves (Fig. 27, p. 79). An infection in the mouth, pharynx or maxillary sinus destined to spread toward the skull travels primarily through the pharyngeal wall or the posterior wall of the maxillary sinus to enter the anterior compartment of the pharyngomaxillary space (p. 79). In the anterior compartment the infection may cause lymphadenitis, abscess or phlegmon. An abscess

of the pharyngomaxillary space (Fig. 54) tends to encapsulation, but a phlegmon definitely tends to spread in one of three directions. (1) It may spread from the anterior to the posterior compartment of the pharyngomaxillary space, then down along the sheaths of the great blood vessels, causing thrombophlebitis of

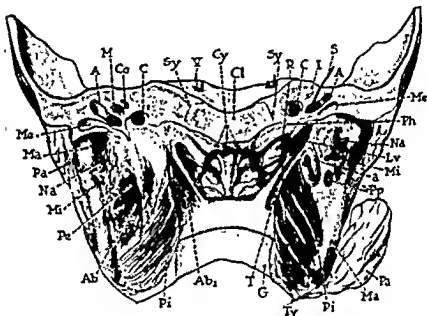


FIG. 54—Abscesses (*Ab* and *Ab₁*) in upper portion of pharyngomaxillary space. *A*, antrum nasutideum, *M*, middle ear, *Co*, cochlea, *C*, internal carotid artery, *Sy*, petro-occipital sychondrosis, *V*, trigeminal nerve, *Gy*, small cysts in mucosa of roof of pharynx, *Cl*, clivus, *I*, long capsule of cochlea, *S*, probe passing through tube and middle ear, *Me*, external auditory meatus, *Ph*, cut through mucosa of posterior wall of pharynx, *R*, groove of Rosenmueller, *Na*, alveolar nerve, *Lv*, levator vel palatini, *Mi*, internal maxillary artery, *u*, localized inflammation in pharyngomaxillary space, *Pp*, veins of pterygoid plexus, *T*, tensor vel palatini, *G*, soft palate, *Fe*, pterygoid external muscle.

the jugular vein, mediastinitis, edema of the larynx, pleuritis and metastases. This type is not uncommon. (2) It may spread along the styloglossus muscle to the floor of the mouth, eventually causing osteomyelitis of the mandible. This type is rare. (3) The phlegmon may spread either within the anterior compartment toward the base of the skull or into the posterior compartment.

then upward along the sheaths of the large blood vessels and nerves of the posterior compartment ultimately causing an intracranial complication. This is less common than the first type but not as rare as the second. The principal pathway in the posterior compartment of the pharyngomaxillary space is the internal jugular vein, which may carry the infection up to the lateral sinus and into the petrosal sinus and carotid plexus, from which it may enter the cavernous sinus. In the anterior compartment the principal pathways are the loose connective tissue, the veins of the pterygoid plexus and the sheaths of the third branch of the trigeminal nerve. The veins may carry the infection into the inferior ophthalmic vein or the veins of the foramen ovale or foramen spinosum and thence into the cavernous sinus (Fig. 4, p. 13). The nerve sheaths carry the infection into the subarachnoid space. The loose connective tissue transmits it into the bones of the base of the skull. In all of these instances the phlegmon of the pharyngomaxillary space causes cavernous thrombosis and therefore is primary. When the phlegmon is caused by a cavernous thrombosis, it is secondary.

Petrosal sinuses and carotid plexus.—These blood vessels carry the infection from the temporal bone to the cavernous sinus. The principal ones are the inferior petrosal sinus and the carotid plexus, the superior petrosal sinus being of minor importance. The inferior petrosal sinus is involved when there is jugular bulb thrombosis. Infection of the carotid plexus (Fig. 55) originates in the anterior part of the tympanic cavity near the eustachian tube or in the petrous apex. Infections of the anterior part of the tympanic cavity are not invariably harmless. Theoretically, the usually thin anterior wall of the cavity and the anastomosing blood vessels between the tympanic mucosa and the veins of the carotid plexus should lead frequently to spread of both acute and chronic infections to the carotid plexus. Actually, this is not common, although it is less rare than is assumed by many otologists who contend that infections of the carotid plexus occur only with tuberculosis of the tympanic cavity. Microscopic studies by the author disclosed thrombophlebitis of the carotid plexus in three

cases of cholesteatoma of the tympanic cavity (Fig. 55), one case of tuberculosis of the tympanic cavity (the carotid plexus being infected by cocci, not by Koch's bacilli) and one case of subacute otitis in a diabetic. Other investigators have found infection of the carotid plexus with acute otitis. Consequently, any infection of the tympanic cavity may involve the carotid plexus and eventu-

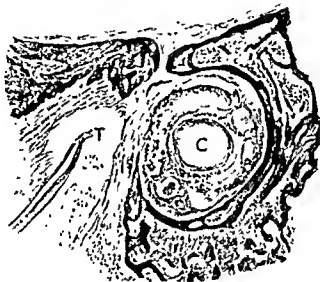


FIG. 55.—Thrombophlebitis of carotid plexus. T eustachian tube, C, internal carotid artery

ally the cavernous sinus. Infections of the petrous apex seldom involve the carotid plexus.

There is a distinct difference between the petrosal sinuses and carotid plexus and the pathways previously discussed. Whereas infections in the orbit or pharyngomaxillary space travel simultaneously along the venous radicles, nerve sheaths and loose connective tissue toward the skull and meninges, infections spreading from the tympanic cavity are limited to the petrosal sinuses and carotid plexus and cause cavernous thrombosis without simultaneous involvement of the meninges and bones of the skull. Furthermore, the walls of these pathways do not contain muscles, consequently, the spread of infection is not assisted by muscular action. In addition, the tympanic type of cavernous thrombosis is

apparently caused by an infection of low grade virulence; otherwise, the infection would probably cause meningitis before arriving at the cavernous sinus. In the tympanic type, also, the orbital infection is invariably secondary. Thus the tympanic type differs from other types of cavernous thrombosis both pathologically and clinically. Some surgeons emphasize that in the tympanic type there is slow occlusion of the cavernous sinus, causing a chronic compensation type of thrombosis, whereas in the other types there is sudden obliteration of the sinus, causing an acute fulminating type of cavernous thrombosis. However, the rate of the occlusion is not a good criterion for differentiation because proof is impossible. More important are the rate of spread toward the sinus and the eventual uniting of infection along the blood vessels with that along the nerves and loose connective tissue. In the tympanic type spread is slow and limited to the two pathways.

Anastomosing blood vessels.—As mentioned on page 24, blood vessels originate in the sphenoid mucosa, run through the walls and drain into the cavernous sinus. An infection of the sphenoid mucosa may cause thrombophlebitis of these anastomosing blood vessels and thrombosis of the cavernous sinus. Although microscope evidence is scanty, this pathogenesis may be inferred in all cases of purulent sphenoiditis and cavernous thrombosis without gross osteomyelitis of the sphenoid. Among 66 cases of rhinogenous, otitic and pharyngeal thrombosis of the cavernous sinus, 17 per cent were of this type.

Meningeal veins.—With extensive thrombophlebitis of the veins of the pia, as may occur in leptomeningitis, the infection occasionally spreads along the meningeal veins to the cavernous sinus. This type of cavernous thrombosis is a terminal condition.

Infection carried into the cavernous sinus along one or more of the venous radicles enters a space which contains several venous channels, the carotid artery, sheaths of several cranial nerves and loose connective tissue (Figs. 2 and 9). Consequently, the infection does not inevitably form a thrombus, as is the case when infection invades the lateral sinus by contiguity, causing inflammation of the sinus wall and necrosis of the endothelium.

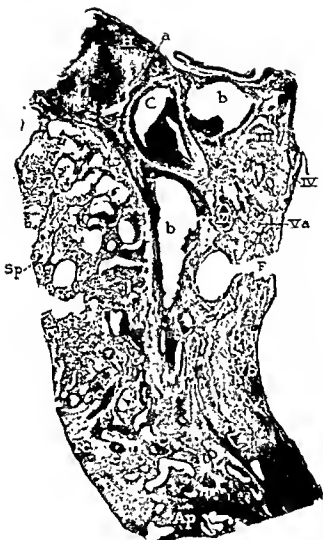


FIG. 56—Thrombosis of left cavernous sinus, oblique section through hypophysis (*H*) internal carotid artery (*C*), body of sphenoid (*Sp*), basal fibro cartilage (*Fc*) and petrous apex (*Ap*) *a*, purulent thrombophlebitis of cavernous spaces, *III*, oculomotor nerve, *IV*, trochlear nerve, *Va*, ophthalmic nerve, *VI*, abducens nerve, *Vb*, maxillary nerve *F*, dural fistula communicating between cavernous sinus and intracranial fossa, *c*, purulent thrombophlebitis of veins between pharyngeal plexus and cavernous sinus

Although thrombus formation is the most frequent result of infection of the cavernous sinus, in some instances the infection primarily involves the loose connective tissue, causing an accumulation of pus while the veins are, to a great extent, simply compressed or necrotic, but not thrombosed (Fig. 56). In either case the condition is called thrombophlebitis and tends to spread into (1) the intercavernous (or circular) sinus, (2) orbit, (3) meninges, (4) pterygoid fossa and pharyngomaxillary space, (5) sphenoid sinus or (6) lateral sinus.

Invasion of the intercavernous sinus is the most important. Virulent infections spread rapidly, sometimes with only a few hours between the clinical manifestation of cavernous thrombosis on the two sides. Frequently it spreads into the orbit, causing thrombophlebitis of the ophthalmic veins and secondary orbital phlegmon. Owing to the connection of the cavernous sinus with the meningeal veins, the infection readily spreads from the sinus into the leptomeninges, causing meningitis or infection of the brain. Spread of the infection toward the pterygoid veins leads to formation of an abscess of the pharyngomaxillary space. This type of spread is often unrecognized because of failure to examine the pharyngomaxillary space in routine autopsies. Along the blood vessels between the sphenoid mucosa and the cavernous sinus the infection may spread into the sphenoid sinus, causing sphenoiditis and osteomyelitis of the walls of the sphenoid. Ultimately the infection may spread along the petrosal sinuses into the lateral sinus. If cavernous thrombophlebitis causes metastases, they are usually found in the lungs.

SYMPTOMATOLOGY

Systemic symptoms.—There is usually severe septicemia. With cavernous thrombosis from infection of the skin of the face, staphylococcal bacteremia is common; with that secondary to infections of the ear, nose or throat, streptococci or staphylococci are occasionally found in the blood. However, blood cultures may be negative even though autopsy reveals multiple pulmonary metastases.

Symptomatically, the septicemia due to cavernous thrombosis does not differ from that caused by infection of other dural sinuses. It is difficult to determine the duration of cavernous thrombosis on the basis of septicemia. If the first manifestation of ocular and orbital symptoms is arbitrarily considered to be the onset of the cavernous thrombosis, the disease usually runs a rapid course. Without adequate treatment, the duration is from several hours to nine days, the average being four days. In ear cases, the thrombosis may last three to four weeks and the ocular and orbital symptoms may fluctuate, even disappearing and re-appearing. The temperature is moderately elevated, with occasional abrupt rises. The picket-fence type of fever becomes definite only shortly before death. These cases are not common. The hypothesis that in such cases there is an aseptic clot in the cavernous sinus is not borne out at autopsy, because the cavernous sinus frequently harbors pus.

General brain symptoms.—These do not differ from those in thrombophlebitis of the other sinuses. Euphoria is a prominent feature. Unless the meninges are involved the patient may insist that he feels well despite high fever. The cerebrospinal fluid is normal or cloudy but does not contain bacteria until just before death. Apparently the cloudiness is due not to the cavernous thrombosis but to the meningitis.

Focal brain symptoms.—These are not present unless the cavernous thrombosis causes brain abscess or softening of the brain.

Ocular and orbital symptoms.—These are considered the principal symptoms. They consist of exophthalmos, edema of the eyelids extending toward the bridge of the nose, cheek or temporal region, chemosis and occasionally hemorrhages in the conjunctiva. The symptoms are unilateral unless the infection spreads from one cavernous sinus to the other. In addition, there is paralysis of the cranial nerves in the orbit, with the abducens and the first branch of the trigeminal nerve most frequently involved. The latter causes stabbing pain in the eye which is increased by pressure on the globe. Occasionally there is corneal

anesthesia and, exceptionally, clouding and necrosis of the cornea. The pupils are usually dilated and fixed or react sluggishly to light. The optic disks are often blurred and may be slightly elevated on the nasal side. The retinal veins are engorged, but definite papilledema occurs in only 10.6 per cent of cases, and in these it appears only one or two days before death. Amaurosis, retinal hemorrhage and general retinal edema, which may pass unnoticed, are even rarer. Papilledema cannot be considered characteristic of cavernous thrombosis and if present, particularly in the initial phase of the illness, frequently indicates some complication other than cavernous thrombosis.

Exophthalmos is due not to the cavernous thrombosis but to the orbital infection. Two observations favor this concept. (1) Obliteration of the cavernous sinus by injection of wax or oil into the sinus does not produce exophthalmos. (2) Among my statistics there are 16 cases (24 per cent) of cavernous thrombosis without ocular or orbital symptoms, even terminally, although autopsy in several revealed involvement of both cavernous sinuses. In only one of the 16 were there fixed pupils. No other ocular or orbital symptoms were present. In eight cases the cavernous thrombosis originated in the tympanic cavity, in six in the pharynx and in two probably in the sphenoid sinus. In contrast, some reported cases presented ocular and orbital symptoms on both sides, although at autopsy only a mural thrombus was found in one cavernous sinus. It is true that exophthalmos occurs in rare instances of aseptic cavernous thrombosis, as it is occasionally seen in debilitated individuals or after injuries. However, even in these cases there is probably orbital involvement, consisting of passive hyperemia or thrombosis of the ophthalmic veins, transudate and perhaps hemorrhages into the orbit. For the same reason transitory ocular and orbital symptoms may occasionally occur with extensive thrombophlebitis of the dural sinuses but without infection of the cavernous sinus, or immediately after ligation of the jugular vein, particularly in children.

The orbital infection caused by cavernous thrombosis varies considerably in extent. In the mildest cases there is only collateral

hyperemia causing a serous exudate into the orbital tissue. An infection of this type resolves spontaneously, so that partial or complete recession of the proptosed eye may occur even in fatal cases. When infection of the cavernous sinus causes an orbital phlegmon, spontaneous cure can hardly be expected, particularly when abscesses develop in the orbit, and the ocular and orbital symptoms will not resolve spontaneously. Since cavernous thrombosis may cause collateral hyperemia as well as phlegmon of the orbit, the intensity of changes in the eye first affected may abate as collateral circulation in the orbit becomes established, while the other eye, involved later, presents progressive exophthalmos. In other cases exophthalmos caused by collateral hyperemia may subside spontaneously, and several days later the orbit may become reinfected, resulting in orbital phlegmon.

Granted that the orbital and ocular symptoms are due to involvement of the orbit and not solely to the cavernous thrombosis, clinical evaluation of the symptoms is not difficult. With paranasal infections, except those of the sphenoid sinus, there is usually a primary orbital phlegmon. Consequently, the ocular and orbital symptoms are striking, although whether the symptoms are due to orbital phlegmon plus cavernous thrombosis or to orbital phlegmon alone is not clear. However, if the septicemia or general brain symptoms are pronounced, involvement of the cavernous sinus may reasonably be assumed. Diagnosis of cavernous thrombosis is definite only when the other eye is involved. In this event the thrombosis has spread through the intercavernous sinuses to the cavernous sinus on the other side and then into the other orbit. Although bilateral exophthalmos is conclusive evidence, bilateral lid edema is not, because unilateral inflammation of the orbit often causes edema of both lids. In cavernous thrombosis of otitic or pharyngeal origin the orbital phlegmon is usually secondary, so that ocular and orbital symptoms are often lacking. Of 12 personal cases of cavernous thrombosis of otitic origin, exophthalmos did not appear in 10, and in two developed only a few days before death. Furthermore, ocular and orbital symptoms in otitic and pharyngeal cavernous thrombosis usually

indicate infection of the orbit plus infection of the cavernous sinus, but in paranasal infections they may be caused by orbital infection alone. Consequently, whereas in rhinogenous infections unilateral orbital and ocular symptoms may or may not be due to cavernous thrombosis, in otitic and pharyngeal infections they almost invariably indicate cavernous thrombosis.

The distinction between primary and secondary orbital phlegmon may seem arbitrary, but it has practical value provided the origin of the cavernous thrombosis is known. This is not always possible. Occasionally cavernous thrombosis considered to be of otitic origin is actually due to unrecognized sphenoiditis or ethmoiditis. In other cases Bezold's mastoiditis causes symptoms of cavernous thrombosis, which is attributed to the otitis when actually it is due to the deep neck abscess which has extended into the pharyngomaxillary space.

A man, aged 45, had recurrent acute otitis on the right side which caused Bezold's mastoiditis, with fever, chills and trismus. On May 19, 1943, the mastoid was operated on and neck abscess incised. Extensive periphlebitis of the lateral sinus was found. The pus contained streptococci. The patient was given sulfonamides, but the neck swelling did not recede. On June 9 the neck abscess was again incised, with the incision extended to the retroauricular incision. The incision was left open. On June 26, the retroauricular incision was reopened. Several sequestrums were found on the sinus and an extradural abscess in the petrous angle was drained. There was marked periphlebitis, but no sign of sinus thrombosis. On June 28 he complained of pain in the right eye, and on July 11 there were slight symptoms of meningitis. On July 12 there was a suggestion of blurring of the optic disk on the right side, and on July 14 there were striking stiffness of the neck, swelling and redness of the left upper eyelid and slight chemosis and paresis of the external rectus muscle. He complained of double vision on looking up. The left cheek was swollen, and there were marked swelling and redness of the right side of the neck, the redness extending along the posterior surface to the left sternocleidomastoid muscle. The left side of the soft palate was swollen. There were no meningeal symptoms. On July 15 the cerebrospinal fluid was sterile but contained 500 polymuclear cells. On July 18 there was pus in the right lateral sinus and right-sided papilledema. He died the next day.

Autopsy revealed basilar meningitis, purulent thrombophlebitis of

the cavernous, superior petrosal and lateral sinuses on both sides, a large extradural abscess in the right posterior cranial fossa, an abscess at the tip of the right temporal lobe, a second abscess in the right occipital lobe and a metastatic abscess in the left lung. In this case there was a severe infection of the right mastoid which did not respond to sulfonamides. Penicillin was not available. Presumably, perforation at the tip of the mastoid caused a neck abscess on the right which, despite surgery and sulfonamides, spread along the posterior surface of the neck to the left side. From here an ascending infection extended into the cavernous sinuses on both sides and thence into the superior petrosal and lateral sinuses. Perhaps radical surgery on the left side of the neck would have arrested the spread of infection.

Nasal symptoms.—Of the paranasal sinuses the sphenoid and posterior ethmoid are the principal offenders. Cavernous thrombosis due to an infection of the frontal sinus, acute osteomyelitis of the skull or maxillary sinusitis of dental origin is not common. Acute sphenoiditis seems particularly prone to cause infections of the cavernous sinuses. Cavernous thrombosis in nurslings from the first week to the ninth month of life is often due to osteomyelitis of the maxilla. The greatest incidence is during the first three weeks after birth. Redness, swelling, purulent discharge, necrosis and sequestration may be present on any or all surfaces of the maxilla. The cause is infection of the tooth buds or maxillary and ethmoid sinuses.

The sphenoiditis destined to cause cavernous thrombosis does not cause unusual symptoms, but there are usually signs of an impending intracranial complication. These include persistent fever, chills, vomiting, delirium, pain in the neck and persistent pain behind the eye. These symptoms and pus in the olfactory fissure and x-ray evidence of a cloudy sphenoid and posterior ethmoid indicate impending complication. Diagnosis is difficult if pansinusitis and infection of the anterior sinuses obscure the infection of the posterior sinuses. The difficulty increases if there is closed empyema of the sphenoid or posterior ethmoid, or both, with no pus and crusts in the olfactory fissure. There may be closed empyema in both acute and chronic infections. In the absence of other symptoms, such

as headache, anosmia and pharyngitis, the sphenoiditis may remain unrecognized even after the bony walls of the sinus are involved. This latent type of sphenoiditis may flare up with an infection of the ear or the tonsils or after a minor nasal operation, particularly resection of the nasal septum. Frequently the aural or tonsillar infection is treated until death, and autopsy reveals that sphenoiditis caused cavernous thrombosis or meningitis, or both. Therefore x-ray examination of the posterior sinuses should be made in every case of cavernous thrombosis unless it has originated in an infection of the skin. If the sphenoid infection has traveled into the cavernous sinus and proper treatment is not instituted, the cavernous thrombosis is fatal in five to 18 days.

Pharyngeal symptoms.—A phlegmon of the pharyngomaxillary space is a potential source of infection of the cavernous sinus. Although the phlegmon spreads along the great blood vessels of the neck toward the mediastinum more often than toward the meninges (p. 220), I venture the opinion that an ascending extension of the infection is frequently concealed by the more conspicuous spread toward the mediastinum. However, the extension is often exclusively an ascending one, and its early recognition is of utmost importance in view of the unfavorable outlook when the infection becomes intracranial.

All infections of the pharynx and mouth may eventually travel into the pharyngomaxillary space. If the pharyngomaxillary infection is due to peritonsillitis, as occurs in 1-2 per cent of all acute inflammations of the tonsils, the following symptoms indicate ascending extension of the phlegmon. (1) Irritation of the trigeminal nerve, particularly the third branch, causes pain in the teeth and eye and eventually typical trifacial neuralgia. Some patients complain of headache radiating to the temporal area, ear or occiput. It is not necessarily a manifestation of an initial phase of basilar meningitis, but may be due to involvement of the auriculotemporal nerve, which leaves the third branch of the trigeminal below the foramen ovale. Headache is not as common as one would expect and often appears only in a late stage. (2) Ear symptoms, due to acute catarrh of the tympanic cavity on

the involved side, include pain in the ear, tinnitus and eventually diminution of hearing. (3) Trismus is highly suggestive, when present, even though the peritonsillar infection is subsiding. (4) The tonsils and palatal arch bulge into the mouth. (5) Swelling of the lateral pharyngeal wall extends toward the base of the skull. Pharyngeal swelling may also be caused by purulent exudate extending down from a cavernous thrombosis of otitic or rhinogenous origin into the pharyngomaxillary space. (6) There may be swelling in the region of the parotid gland. (7) Swelling of the temporal region is caused by thrombophlebitis of the temporal veins or the pterygoid plexus or by an abscess in the temporal muscle. It never extends below the zygomatic arch.

Infection ascending from the pharynx has either a fulminating course of four to eight days or a delayed course of nine to 30 days. In the latter type the symptoms of cavernous thrombosis appear only in the terminal stage.

Infections of the pharyngomaxillary space are frequently unrecognized. The reason is obvious. The patient is seriously ill, and the profound septicemia so engages the surgeon's attention that close examination of the throat is performed only if there is swelling of the neck, if the patient complains of difficulty in swallowing or if he is spitting blood. Hence a moderate swelling of the lateral pharyngeal wall or a displacement of the tonsil is often overlooked, especially after trismus renders examination of the throat difficult.

A man, aged 45, had acute otitis on the left, with fever. After paracentesis the drum membrane became gray, but there were chills, spiking temperature and slight edema of the left mastoid process. A simple mastoid operation disclosed no pathology in the mastoid or lateral sinus. Nevertheless, the jugular vein was ligated. Two days after operation there were exophthalmos, chemosis and abducens paralysis on the left side, but no meningeal symptoms. Based on incorrect interpretation of an x-ray film, a Strett operation (p. 126) was performed, but no pus was discovered at the petrous apex. Sulfonamides and penicillin were not available. At autopsy cavernous thrombosis was discovered on both sides, but no meningitis. In the left temporal bone, the jugular bulb, carotid plexus and apex were normal.

Microscopic examination of the left cavernous sinus revealed an accumulation of pus which had extended toward the sella turcica and the greater wing of the sphenoid bone. Toward the carotid plexus the inflammation decreased, and there was practically none in the carotid canal of the temporal bone. However, there was definite thrombophlebitis of the vesalian veins, which run through the basilar fibrocartilage and create an anastomosis between the cavernous sinus and the pterygoid plexus in the pharyngomaxillary space.

The pathology in this case is not clear, yet it can be stated definitely that the cavernous thrombosis was not of otitic origin. Therefore the surgery performed was unnecessary. Actually, jugular ligation probably encouraged formation of the cavernous thrombosis. Microscopic examination indicated that thorough examination of the pharynx and mouth probably would have established the correct diagnosis.

Otitic symptoms.—Cavernous thrombosis can be caused by any type of otitis. My statistics show that it was caused by acute otitis in 16.5 per cent and by chronic otitis in 21 per cent of cases. In one case the thrombosis originated in tuberculous otitis with secondary streptococcic infection. Not infrequently cavernous thrombosis develops after a mastoid operation, either in one or several days or after six to eight weeks. In all such cases convalescence after the mastoid operation is disturbed. In infants the symptoms of cavernous thrombosis may be noted immediately after jugular ligation.

Infections of the tympanic cavity have a greater tendency to spread toward the lateral sinus or jugular bulb than toward the cavernous sinus. Usually there is extensive thrombophlebitis of the lateral sinus and jugular bulb prior to involvement of the cavernous sinus. Obviously the symptoms of thrombophlebitis of the lateral sinus and jugular bulb obscure those of cavernous thrombosis, since in cavernous thrombosis of otitic origin the orbital infection is always secondary. When cavernous thrombosis of nasal or pharyngeal origin extends into the lateral sinus, the ocular and orbital symptoms are more striking, and the tympanic cavity is normal, although the skin over the mastoid process may be edematous.

Despite lack of clinical symptoms which stigmatize otitis as

a potential source of cavernous thrombosis, the possibility of otitic cavernous thrombosis must be kept in mind after operations for thrombophlebitis of the lateral sinus. If the septicemia persists, the surgeon usually attributes it to thrombophlebitis of the jugular bulb or progressive thrombophlebitis of the lateral sinus. However, the septicemia may be due to thrombophlebitis of the inferior petrosal sinus or carotid plexus which is advancing toward the cavernous sinus.

PROGNOSIS

Theoretically, there are two reasons why the prognosis for cavernous thrombosis should be more favorable than that for lateral sinus thrombophlebitis. First, in the cavernous sinus the considerable amount of connective tissue should assist in localizing the infection, as it does in the subarachnoid space and the perilymphatic space of the inner ear. Hence, spontaneous cure should be more frequent in cavernous thrombosis than in lateral sinus thrombophlebitis. Second, with infections of the cavernous sinus there is usually only thrombosis, not thrombophlebitis. That is, the wall is not involved, or is involved only to a minor degree, except in cavernous thrombosis originating in osteomyelitis of the sphenoid. Considering that in lateral sinus thrombosis failure of treatment and further complications are often due to progressive inflammation of the sinus wall, one should expect more favorable conditions in infections of the cavernous sinus. Unfortunately, theoretical arguments are not borne out in clinical experience. The prognosis for cavernous thrombosis is not good. There are two reasons for the discrepancy between theory and practice. First, the carotid artery running through the cavernous sinus acts as a dynamic force driving the infection in all directions, particularly into the meninges, lungs and, occasionally, the brain. Furthermore, the cavernous thrombosis is nearly always preceded by infection of the tributary veins—the ophthalmic veins, the veins of the pterygoid plexus, the petrosal sinuses and the carotid plexus. Hence, in the presence of cavernous thrombosis, one may assume that there is widespread infection of the veins of

the skull. The exception is cavernous thrombosis due to sphenoiditis, in which the sinus wall does become involved. However, in such cases there is almost simultaneous meningeal infection, so that the outlook is no more favorable.

To sum up, the prognosis for cavernous thrombosis is serious not because of the thrombosis itself but because of the severe infection which precedes it and the rapid extension of infection which follows. If for any reason the infection does not spread, spontaneous recovery may ensue. In fact, spontaneous cure has been reported for all types of cavernous thrombosis. In 45 cases of all types of cavernous thrombosis, mortality was 69 per cent. There is no doubt that chemotherapy will materially diminish the mortality rate provided correct diagnosis regarding the origin of the cavernous thrombosis is made.

TREATMENT

There is no standardized treatment of cavernous thrombosis. It seems to be established that chemotherapy is superior to surgery of any type. Penicillin, sulfonamides and bacteriophage are the preparations of choice. Penicillin and sulfonamides must be given in maximal dosage. Some surgeons administer 30 gr. (2 Gm.) of sulfathiazole plus 15 gr. of sodium bicarbonate every four hours, i.e., a total of 180 gr. (12 Gm.) in 24 hours, for 14 days. Then the dose is gradually reduced until the patient is discharged on a dose of 1 Gm. three times daily for at least two weeks. Usually a smaller dose is used, with 8-12 Gm. the first day and 4-8 Gm. daily thereafter for 10-14 days.

Staphylococcus bacteriophage is given intravenously in a series of regularly spaced, increasing doses until a chill results, or until a total of 500-1,000 cc. has been administered the first day. Subsequently, bacteriophage is injected at least twice daily until the patient appears normal, and then once daily for two weeks. It is then administered once or twice a week for six months to prevent sequelae, such as brain abscess.

Heparin or coumarin should be added, and fluids and blood transfusions should be given according to the requirements of

the individual patient. *Streptococcus antiserum*, intracarotid injections of rivanol, *staphylococcus antitoxin* and other antiseptic preparations have been used successfully but are no longer routinely employed. This type of treatment should be used, when indicated, in conjunction with surgical procedures on the mastoid, paranasal sinuses or pharyngomaxillary space.

The surgical treatment of cavernous thrombosis includes (1) procedures designed to combat the infection indirectly, but not to attack the cavernous sinus proper, and (2) direct procedures designed to drain the cavernous sinus.

1. The principal indirect method is the ligation of the common carotid or internal carotid artery to arrest pulsations of the artery within the cavernous sinus and to place the sinus at rest. Although a few reported cases indicate that the procedure was successful, it did not become popular for various reasons. The procedure is hazardous because ligation of the common or internal carotid artery may cause damage to the brain, particularly when the cerebral blood circulation has not been prepared by systematic digital compression of the carotid artery before ligation. Furthermore, experience with arteriovenous aneurysms of the cavernous sinus proves that carotid ligation does not prevent recurrences of the aneurysm. There are free anastomoses between the internal and external carotid systems through the circle of Willis as well as through the orbital branches of the ophthalmic artery, which communicate with the ophthalmic artery on the opposite side and with the orbital, facial, temporal and meningeal branches of the external carotid. These anastomoses may, in a few hours, carry blood into the internal carotid which was shut off by ligation. For these reasons I do not ligate the carotid artery in cases of cavernous thrombosis.

Other indirect procedures include drainage of the superior petrosal sinus within the temporal bone, drainage of the jugular bulb and subsequent aspiration of the thrombus from the inferior petrosal sinus and drainage of the petrous apex. All have been used successfully in individual cases. They should be performed when there are clinical indications and the patient's general con-

dition permits surgery. They cannot, however, be considered standard operations.

Of practical importance are incisions of the orbit. These are based on a sound surgical principle in primary, but not in secondary, orbital phlegmon. Nevertheless, the orbit has been incised in both types of orbital phlegmon, with reported success when pus was evacuated from the orbit. Usually, however, no pus was discovered because the infection had involved only the orbital veins or the abscess had been localized at the orbital apex near the optic foramen. In these cases it is doubtful that incisions of the orbit were beneficial, particularly when access was gained by preparation of a Kroenlein flap, which must be considered a major operation.

Despite all risks, there would be no substantial objection to incision of the orbit if a diagnosis of frank pus could be made prior to the operation. This is possible only if the abscess extends forward to the conjunctiva, and in this event a spontaneous rupture often occurs. These instances are, however, rare. Therefore incision of the orbit is apparently advisable if chemotherapy fails or if amaurosis impends. Postponement of the operation is not dangerous because eight days or more are required for frank pus to form in a primary orbital phlegmon and two weeks or more in a secondary phlegmon.

2. The principal direct approaches are through the orbit, through the sphenoid and through the middle cranial fossa.

In the approach through the orbit, performed by the technic of Mosher, the globe is removed, the orbit exenterated and the ophthalmic artery ligated. The next step is to separate the perosteum from the orbital surface of the greater wing of the sphenoid and to identify the outer end of the sphenoid fissure. With the chisel placed vertically, a cut is made through the orbital plate of the greater wing and enlarged outward 0.5 cm. The lower edge of the bony window is brought flush with the floor of the orbit. The dura is then elevated from the floor of the middle cranial fossa. After exposure of the outer wall of the cavernous sinus and separation of the dura from it for 1 cm., a

blunt knife is placed against the outer wall of the sinus on a level with the floor of the orbit and the knife blade is carried forward toward the body of the sphenoid until it is arrested by bone. Through this incision into the cavernous sinus a small curet can be inserted to the openings of the superior and inferior petrosal sinuses.

The approach through the sphenoid sinus consists of removal of the ethmoid labyrinth, middle turbinates and anterior sphenoid wall. A curet is pushed through the roof of the sphenoid sinus close to its junction with the external wall. The blunt end of the curet pushes the carotid artery aside without damage, and by rotation of the spoon in a forward direction away from the artery, its edge catches bone and a hole of some size can be made, leading directly into the cavernous sinus.

The approach through the middle cranial fossa is the same as the typical approach to the gasserian ganglion.

These direct methods are extensive and sanguinary procedures which patients with profound septicemia can seldom endure. All that can be accomplished is incision of the sinus and, occasionally, partial removal of a clot. Experience in lateral sinus thrombophlebitis has proved that simple incisions of the sinus with or without partial removal of the clot may be successful in favorable cases without tendency to spread. In progressive cases these procedures are not effective. In view of the facts that in cavernous thrombosis the tendency to spread is marked and that the spongelike structure of the sinus will scarcely permit of free surgical drainage, the direct surgical approach cannot be advised as a routine procedure.

THROMBOSIS OF THE SUPERIOR LONGITUDINAL SINUS PATHOLOGY

This section deals with thrombosis of the superior longitudinal sinus caused by infections of the ear and paranasal sinuses.

As in the cavernous sinus, infection of the superior longitudinal sinus occurs more frequently by continuity than by contiguity. Hence, the term thrombosis, not thrombophlebitis, is ap-

plied. The frontal sinus is the principal offender; although a few recorded cases indicate that the maxillary or ethmoid sinus may be the source of infection. An infection of the frontal sinus may involve the superior longitudinal sinus either by way of anastomosing blood vessels or by causing osteomyelitis and extradural abscess. It is likely, although not proved, that in infections of the frontal sinus the many small veins which carry blood from the mucosa of the frontal sinus into the superior longitudinal sinus (Fig. 5) become involved with thrombophlebitis and carry septic material into the sinus, causing septicemia. Actual thrombus in the superior longitudinal sinus, continuous with thrombophlebitis of the small anastomosing veins, is not common. This is in accordance with the findings in osteophlebitis pyemia (p. 148), which also causes septicemia but seldom thrombophlebitis of the lateral sinus. Thrombosis of the superior longitudinal sinus is also uncommon with acute osteomyelitis of the skull originating in the paranasal sinuses. Some writers believe that it occurs in about 25 per cent of cases, particularly in those with subdural empyema from the osteomyelitis. This coincides with my experience. It is likely that thrombosis of the superior longitudinal sinus would be reported more frequently if the anterior portion of the sinus were always examined at autopsy.

If the ear is the source of infection, thrombosis of the superior longitudinal sinus is simply a continuation of thrombophlebitis of the lateral sinus, particularly with involvement of the right lateral sinus, which usually anastomoses freely with the superior longitudinal sinus (p. 16). In these instances the infection of the sinus wall does not necessarily extend from the lateral to the superior longitudinal sinus. In some a marasmic thrombus of the longitudinal sinus apparently joins the septic thrombophlebitis of the lateral sinus and becomes infected secondarily. Favoring this concept is the frequent observation of marasmic thrombi in the longitudinal sinus.

Apparently thrombosis of the longitudinal sinus has a different course in rhinogenous and in otogenous infections. Rhinogenous thrombosis is probably caused by a particularly virulent infection;

otherwise it would occur more often with frontal sinusitis, a rather common type of infection. Because of the virulence, the course is rapid, and autopsy reveals not only thrombosis of the longitudinal sinus but cavernous thrombosis, external and internal pachymeningitis and, eventually, meningitis, encephalitis or brain abscess.

In a personal case there was a large subdural empyema between the occipital lobes although the infection had originated in the frontal sinus. Apparently the purulent exudate had gravitated toward the occiput, owing to the recumbent position of the patient.

In otogenous infections the lateral sinus is always primarily involved. From the lateral sinus the infection slowly creeps into the longitudinal sinus, and several weeks or even months may elapse before a thrombus forms in the longitudinal sinus. It can reasonably be assumed that the infection is not particularly virulent. If it were, the lateral sinus thrombophlebitis would cause meningitis before arriving at the longitudinal sinus, or the infection would spread along the meningeal and cerebral veins from the longitudinal sinus into the meninges and brain. This does not occur. For this reason the outlook is apparently more favorable than for rhinogenous cases and, as in cavernous thrombosis, the course is more protracted than in rhinogenous cases, which usually run a fulminating course.

SYMPTOMATOLOGY

In many cases thrombosis of the superior longitudinal sinus does not cause significant symptoms. In others, a tentative diagnosis can be made.

Systemic symptoms.—The symptoms of septicemia are usually striking, particularly in cases of rhinogenous origin. In cases of otitic origin the symptoms have minor diagnostic value since they may be caused by either thrombophlebitis of the lateral sinus or longitudinal thrombosis. However, in infection of the frontal sinus or acute osteomyelitis of the skull the appearance of symptoms of septicemia without those of cavernous thrombosis justifies a tentative diagnosis of thrombosis of the superior longi-

tudinal sinus. It should be kept in mind that a paranasal infection as well as an acute osteomyelitis may, rarely, cause septicemia without involvement of the dural sinuses.

Thrombosis of the superior longitudinal sinus, particularly of otitic origin, does not always cause the picket-fence type of fever characteristic of septicemia. The temperature may be only moderately raised, and fluctuations are not conclusive. High fever may not be present until terminal meningitis sets in. In other cases of otitic origin periods of normal temperature are followed by periods of high fever, the cycle being repeated twice or even three times. In a few instances streptococci or staphylococci have been discovered in the blood.

General brain symptoms.—These are not uncommon in children but seldom occur in adults unless the meninges or brain is invaded. Papilledema seems to occur more frequently in thrombosis of the superior longitudinal sinus than in thrombophlebitis of the lateral sinus.

Recently the opinion was advanced that the clinical syndrome of otitic hydrocephalus (p. 294) is symptomatic of longitudinal sinus thrombosis. The hydrocephalus is believed to be caused by inactivation of a sufficiently large proportion of arachnoid villi and by failure of normal absorption of cerebrospinal fluid. However, there are many reported cases, confirmed at autopsy, which presented neither internal nor external hydrocephalus. Other writers state that in the absence of meningitis, the early onset of apathy and stupor in a patient with evidence of thrombosis of the lateral sinus indicates infectious longitudinal sinus thrombosis, especially if there are, in addition, papilledema and convulsions.

Focal brain symptoms.—Many focal brain symptoms have been noted, including convulsions, hemiplegia rapidly ascending from the foot to the face, aphasia and Babinski's reflex. Since these symptoms are not always present, they probably occur only when the thrombosis extends into the superior parietal veins and thus interferes with the functions of the motor cortex.

Local extracranial symptoms.—These symptoms apparently have great practical value if they develop in the early phase of

the infection. The superior longitudinal sinus anastomoses through the emissaria with the veins of the scalp (p. 16). With the sinus thrombosed, the extracranial veins become involved either by passive hyperemia or by infection. This causes, particularly in children, dilatation of the veins of the scalp, edema of the scalp which may extend to the forehead or tender swelling of the skin always localized near the midline, either at the vertex or at the posterior portion of the longitudinal sinus. The swelling may contain pus, granulations or blood. A similar swelling at the same site may occur in acute osteomyelitis without longitudinal sinus thrombosis. Differential diagnosis is difficult because longitudinal sinus thrombosis may cause secondary osteomyelitis which on the x-ray film simulates acute osteomyelitis of rhinogenous origin. However, differentiation is not highly important because in either condition the scalp abscess must be drained.

Ear symptoms.—Thrombosis of the superior longitudinal sinus may be associated with either acute or chronic otitis. In several cases mucosis otitis (p. 103) was the cause. Therefore there is no ear symptom indicating longitudinal thrombosis. Lateral sinus thrombophlebitis with an extremely chronic course, over several months, is suggestive.

Nasal symptoms.—Severe infections of the frontal sinus or ethmoid, or both, are usual. Chronic sinusitis seems to cause longitudinal thrombosis more frequently than does acute sinusitis. However, the nasal findings are not significant. They become more important when associated with septicemia, pain in the vertex and swelling of the scalp. Longitudinal sinus thrombosis has been noted after minor endonasal operations such as resection of the nasal septum and middle turbinate and cauterization of the middle nasal meatus, particularly in the presence of acute infection of the frontal sinus.

PROGNOSIS

The prognosis for rhinogenous longitudinal sinus thrombosis is unfavorable. Despite radical surgery the infection usually causes fatal meningitis. The effect of chemotherapy is not definitely es-

tablished. The outlook is more favorable for the otitic type. In fact, in several cases on record cure was accomplished by surgery without chemotherapy and even without complete removal of the thrombus.

TREATMENT

As in cavernous thrombosis, there is no standardized treatment of thrombosis of the superior longitudinal sinus. Furthermore, the condition is so uncommon that a surgeon scarcely can gain enough experience for proper evaluation of treatment. The following general comments may be helpful. In all instances of rhinogenous longitudinal sinus thrombosis chemotherapy and blood transfusions must be tried first. Surgery should be undertaken if conservative treatment fails, if the infection of the paranasal sinuses requires surgery or if there is osteomyelitis, either progressive or self-limiting. If there is rhinogenous osteomyelitis and the longitudinal sinus must be exposed near the site of the infection, the bone must be removed until normal bone is reached regardless of whether the dura is normal or not. This differs from the procedure in lateral sinus thrombophlebitis, in which the brain is protected by the thick layer of neck muscles when a large part of the occipital squama is removed, whereas in longitudinal sinus thrombosis the brain is not protected when bone is removed. Furthermore, in lateral sinus thrombophlebitis the sinus must be incised and the thrombus removed, but in longitudinal sinus thrombosis it is often impossible to differentiate the sinus when there is marked external pachymeningitis beneath the frontal squama. Therefore all that can be done is to expose the external pachymeningitis by sequestrectomy or removal of infected bone and leave the rest to chemotherapy and other conservative measures. After the operation the patient's head should be raised to prevent gravitation of pus toward the occiput.

In longitudinal sinus thrombosis of otitic origin the operation performed for thrombophlebitis of the lateral sinus must be continued toward the torcular and, eventually, toward the vault of the skull. Extensive removal of the bone of the vault is neither

advisable nor necessary. The thrombus in the longitudinal sinus is likely to be marasmic, so it is not necessary to extend the operation so far that free hemorrhage occurs from the peripheral part of the sinus. With subperiosteal abscesses at the vertex, the bone must be removed at the site of the abscess and the rest of the longitudinal sinus left alone.

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Inflammatory Diseases of the Leptomeninges

LEPTOMENINGITIS

PATHOGENESIS AND PATHOLOGY OF PURULENT MENINGITIS

INFECTION MAY cause purulent meningitis by three mechanisms: (1) infection by contiguity, (2) infection by continuity, and (3) infection by injury.

Infection by contiguity.—This is the common pathway in otitic meningitis and less usual in rhinogenous and pharyngeal meningitis. Spread by contiguity (Fig. 57) primarily causes an infection of the dura and localized external pachymeningitis which may involve a small or large area of the adjacent dura. The infection travels through the dura and causes internal pachymeningitis which by contiguity carries the infection into the leptomeninges. In the subarachnoid space the meningitis remains localized for a time but ultimately becomes diffuse if proper treatment is not instituted. This type of infection occurs particularly with chronic infections of the tympanic cavity or paranasal sinuses. With acute inflammation, the meningitis usually becomes manifest in the late stage of otitis or sinusitis. This is illustrated by cases of meningitis which develop six to eight weeks after the onset of acute otitis, particularly in aged individuals.

In all cases of meningitis caused by infection by contiguity the following observations are of practical importance. (1) There

is usually one portal or one principal portal of invasion. (2) At the portal of entry there are osteitis and localized infection of the dura. (3) The portal of entry can usually be drained by sur-

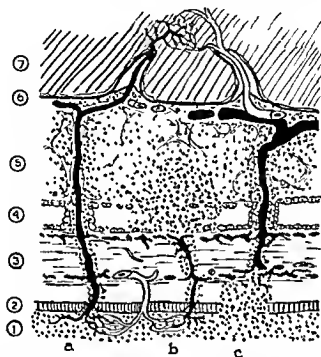


FIG. 57.—Leptomeningitis due to *a*, infection by continuity, *b*, dural infection by continuity and arachnoid infection by contiguity, and *c*, dural infection by contiguity and arachnoid infection by continuity. 1, inflamed mucosa, 2, bone, 3, dura, 4, subdural space, 5, subarachnoid space, 6, pia, 7, brain.

gery. (4) If the portal of entry is properly drained, extension of the infection is usually checked.

A special type of infection by contiguity occurs when a brain herniation is involved. As previously mentioned, brain herniations are occasionally noted in the tegmen tympani, marrow spaces surrounding the eustachian tube and the cribriform plate. In several cases on record an acute infection of a brain herniation was believed to have caused fulminating meningitis. Such cases are not common.

Infection by continuity.—The infection may travel along spe-

cific pathways which run directly into the subarachnoid space without involvement of the dura, or it may travel along the anastomosing blood vessels toward the dura. Infections which travel along the internal auditory meatus (Fig. 16), the arachnoid sheaths of the olfactory bundles (Fig. 58) and, in rare instances, along a patent craniopharyngeal canal (p. 78) enter the subarachnoid space directly, causing leptomeningitis without gross involvement of the dura. The cochlear aqueduct, also a specific pathway, plays a minor role; it more frequently allows an infection to travel from the meninges to the inner ear, as in epidemic cerebrospinal meningitis, than vice versa.

Common pathways are the veins which run between the meninges and the pterygoid plexus and, particularly, the anastomosing blood vessels (Fig. 57), which are well developed in early childhood and, though supposedly becoming obliterated in adults, frequently persist in the walls of the paranasal sinuses (Fig. 5) and incompletely pneumatized temporal bones. These blood vessels are considered responsible for the meningitis in the early phase of acute otitis or acute sinus infections, particularly in children and young persons. In fact, microscopic examinations have demonstrated septic thrombosis or periphlebitis of the anastomosing blood vessels in such cases, and bacteria have been found in them. These examinations reveal only the pathway of infection from the mucosa to the dura; that from the dura to the subarachnoid space is largely hypothetical. One may infer that the thrombophlebitis of the blood vessels advances from the external

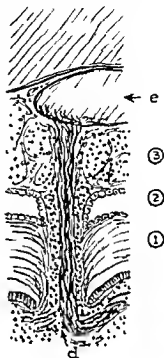


FIG 58 — Inflammation traveling from nasal mucosa (d) along sheaths of olfactory fibers into subarachnoid space (3). 1, dura, 2, subdural space, e, olfactory bulb.

into the internal network of the dura and thence crosses the subdural space along the pial blood vessels which anastomose with the internal network of the dura. By this means the infection might travel from the mucosa to the pia-arachnoid without gross involvement of the dura.

Two arguments might be raised against this concept. Infants, in whom there is the greatest number of anastomosing blood vessels, should show an extremely high incidence of purulent meningitis. In fact meningitis, both purulent and serous, is the most frequent intracranial complication in infants, but the incidence, particularly of purulent meningitis, is slight in comparison with that of otitis and rhinitis in this group. However, in infants the eustachian tube provides natural drainage of purulent exudate in the tympanic cavity and perforation of the drum membrane provides additional drainage, so that retention of pus in the tympanic cavity is less likely in infants than in adults. Since in acute infections retention of pus is one of the most important causative factors, it is conceivable that the incidence of purulent meningitis of otitic origin is not as high in early childhood as one would assume because of the number of anastomosing blood vessels. Moreover, because of the complete or partial absence of pneumatic cells in the infant temporal bone, the acute infection of the mucosa is not as widespread as in the well pneumatized adult temporal bone. Therefore the comparatively low incidence of purulent meningitis in youngsters is not a valid argument against the importance of the anastomosing blood vessels in the pathogenesis of meningitis.

The second argument against the importance of the anastomosing blood vessels assumes that these vessels are capillaries which cannot carry sufficient bacteria to cause meningitis. This, however, is counterbalanced by the multitude of capillaries involved. In temporal bones that are not well pneumatized, the capillaries carry the infection simultaneously into the middle and posterior cranial fossae and toward the jugular bulb. For this reason there is not just one portal of entry, as in infections by contiguity, but rather several sites at which the meninges are at-

tacked by the micro-organisms. Therefore localized meningitis does not develop before diffuse meningitis. This renders surgery less effective. In fact, all that can be achieved by surgery is drainage of the purulent focus in the temporal bone or paranasal sinuses; surgical drainage of the pathways of infection is impossible.

Although the importance of the anastomosing blood vessels should not be questioned, the meningeal infection is not, as a rule, caused exclusively by these blood vessels. Microscopic examinations reveal, in addition, minute spots of osteitis which favor infection by contiguity. Congenital dehiscences of the bone also occasionally have a role, although their importance is frequently exaggerated. In rhinogenous meningitis microscopic examination may reveal septic thrombophlebitis of the anastomosing blood vessels in the posterior wall of the frontal sinus or roof of the ethmoid and an additional infection of the fila olfactoria and osteomyelitis of the cribriform plate. However, these findings are coincidental (except infections along the olfactory bundles), and the principal pathways are the anastomosing blood vessels.

Infection by injury.—Only injuries caused by surgical operations are considered. Postoperative meningitis occurs with both nasal and nasal operations. In the ear, luxation of the stapes and injuries of the semicircular canals and meninges are potential sources of meningitis.

Luxation of the stapes may occur with paracentesis of the drum membrane, extraction of polyps in the tympanic cavity and radical mastoid operations. Luxation causes severe dizziness and spontaneous nystagmus. These symptoms diminish after several hours, and the future course depends on whether or not the inner ear becomes involved. If infection of the inner ear occurs, it usually appears within 12-48 hours after injury. Then dizziness and coarse nystagmus to the other side reappear, the patient is deaf on the involved side and fatal fulminating meningitis often follows in a few days. The fulminating course is noted particularly when luxation occurs during a radical mastoid operation. In these cases the surgeon is not aware of having caused a stapes luxation

because, with the patient under general anesthesia, neither dizziness nor nystagmus is apparent. Hence the surgeon packs the mastoid and tympanic cavity with gauze, encouraging infection of the inner ear. The best means of preventing spread of infection into the labyrinth are loose drainage of the tympanomastoid cavity, immobilization of the patient's head and, particularly, adequate postoperative chemotherapy. However, even without chemotherapy, stapes luxation caused by radical mastoid operation is not necessarily followed by purulent labyrinthitis and meningitis.

A bilateral radical mastoid operation was done on a girl aged 4. Following surgery she became deaf and mute, but the caloric test revealed normal excitability of both labyrinths. When she was 25 she died of septicemia subsequent to tonsillitis. Microscopic examination revealed a fissure of the foot plate on one side. On the other side the stapes was turned 180 degrees, the head being directed toward the inner ear. This severe injury to the stapes had not been followed by purulent labyrinthitis, although chemotherapy was not available at the time.

In general, the outcome of these injuries depends largely on the type and virulence of the infection in the tympanic cavity.

The semicircular canals may be injured during a simple or radical mastoid operation. Usually the horizontal semicircular canal is injured, rarely the posterior and exceptionally the superior semicircular canal. When the horizontal semicircular canal is injured the patient presents dizziness, nausea, nystagmus to the other side and usually facial paralysis immediately after waking from the anesthesia. The labyrinthine symptoms gradually disappear in several weeks, and the facial paralysis improves after several weeks or months, occasionally after one to two years, if the nerve was not severed. The final results are deafness and non-excitability of the injured labyrinth. Meningitis is not common, although in cases of mucous otitis it may develop several days or weeks after injury.

Injuries of the dura may occur during any operation on the temporal bone. They occur particularly when the dura of the mid-

dle cranial fossa bulges deeply into the antrum and the dura is elevated from the roof of the eustachian tube or the posterior surface of the petrous bone, where it is firmly adherent to the bone and can easily be torn. Serious injury may be caused when the dura is exposed and a small bone chip with sharp edges glides between the dura and the remaining bone plate. When the dura alone is injured only a small amount of cerebrospinal fluid escapes because the subdural space does not contain much fluid. If a large amount of fluid is noted it is likely that the arachnoid is also injured. Injury of the dura followed by meningitis requires a guarded prognosis. However, this complication is not common, the meningeal infection usually remaining localized even without chemotherapy. Some surgeons advise enlargement of the opening in the dura by an incision so that the brain may bulge into the opening and close it tightly. Such an incision should be made if the dura is injured by a sharp curet or drill, which causes wounds with ragged margins and often penetrates deeply, occasionally injuring even the cerebral cortex. A gouge or chisel causes a wound with smooth margins which soon closes, but a packing of the wound must be strictly avoided, as after injuries of the labyrinth. The outcome of these injuries depends principally on the virulence of the infection in the tympanic cavity but, in general, is favorable.

A peculiar type of surgical injury of the dura deserves mention. If a simple mastoid operation advances toward the perisinus cells in the superior petrosal angle there is occasionally observed a pneumatic cell in front of, or behind, the lateral sinus containing what seems to be granulations originating from the cell wall. From these granulations a fluid escapes which has the appearance of water mixed with blood. After careful removal of the granulations a small area of the dura is noted, and from this exposed area cerebrospinal fluid escapes with pulsating rhythm. If more of the dura is exposed by removal of bone, it is found to be very thin, slightly bulged and bluish, and close to the sinus are one or two holes in the dura through which cerebrospinal fluid escapes. The holes are the size of a pinhead and their mar-

gins sharp. Although no microscopic specimens are available, I believe that these findings are due to arachnoid villi which occur not infrequently on both sides of the lateral sinus (Fig. 12). The villi perforate the dura and bone and thus come in contact with the mucosa of the mastoid cells, or with granulation tissue if there is mastoiditis. During a mastoid operation the mechanical concussion loosens the connection of the villi with the rest of the arachnoid and opens the subarachnoid space, allowing the outflow of cerebrospinal fluid. If the granulation tissue or inflamed mucosa of the mastoid cells is removed, the villi are also removed, no matter how carefully the operation is performed. When the arachnoid villi are removed, the opening in the dura that has allowed protrusion of the villi can be noticed. The dura is always thin in this area and so gives way to cerebrospinal fluid pressure and bulges. The bluish color is due to the underlying blood vessels of the pia-arachnoid. I have seen two cases of this anatomic type. Both patients recovered, although one had additional surgical injury of the sinus and erysipelas. This patient was treated with prontosil, and the other recovered without chemotherapy.

All types of extra- and endonasal operations on the frontal, ethmoid and sphenoid sinuses occasionally cause meningitis. In external operations on frontal and ethmoid sinuses the course after injury of the dura is essentially the same as that after injuries during a mastoid operation. Meningitis after endonasal operations is more common. Some statistics indicate that among the deaths due to paranasal infections over 70 per cent occur after surgery, whereas among deaths due to ear infections only 1.7 per cent are postoperative. The following endonasal operations should be mentioned: resection of a deviated septum, extraction of nasal polyps, endonasal ethmoid and sphenoid operation, resection of the middle turbinate, lavage of the frontal sinus and endonasal frontal sinus operation. Although the incidence of meningitis is small as compared with the frequency of these procedures, almost every rhinologist has met with such complications.

Meningitis subsequent to endonasal surgery may be caused by (1) fracture of the cribriform plate, (2) infection of the

sheaths of the olfactory bundles or (3) acute infection or acute exacerbation of a chronic infection of the paranasal sinuses.

Fractures of the cribriform plate are direct or indirect. They are direct if the surgeon perforates the plate with an instrument, and indirect when the surgical removal or fracture of an osseous plate in the nasal cavity involves the cribriform plate. Direct fractures are most often due to lavage of the frontal sinus, intranasal frontal sinus operation and endonasal ethmoid or sphenoid operations. In frontal sinus operations the injury is due to poor technic whereby the probe, cannula or other instrument is pushed (usually with considerable force) through the cribriform plate instead of into the nasofrontal duct. The result is fulminating meningitis. The following case illustrates the fulminating course and the inadequacy of the sulfonamides in such cases.

A young girl had pansinusitis on the right, with swelling around the right eye. A physician, apparently not well acquainted with the anatomy of the sinuses, attempted to puncture the frontal sinus by pushing a cannula through the frontal process of the right maxilla into the sinus. Immediately after this procedure meningeal symptoms appeared. Sulfonamides were administered and she seemed to improve, but three weeks later, when hospitalized, she was unconscious and there was hemiparesis on the left. The neurosurgeon opened the skull and dura at several sites in the right parietal and occipital areas and discovered pus under the dura. When I saw the patient in consultation the condition seemed hopeless. On the urging of the neurosurgeon, both frontal sinuses were opened. There was a little pus in the sinuses and the posterior walls were normal, but the soft tissue in the area of the right supraorbital arch was necrotic. The following day the patient died. Autopsy revealed meningitis at the convexity of the brain, many thrombosed pial vessels and perforation of the right cribriform plate.

In intranasal frontal sinus operations the hazard is increased, although not all fatalities are recorded. In endonasal ethmoid operations the incidence of postoperative meningitis is comparatively high, 33 per cent of all cases of postoperative meningitis after endonasal surgery having been attributed to ethmoid operations. Consequently, if autopsy discloses defects in the roof of the ethmoid or in the cribriform plate (Fig. 65, p. 312), the diag-

nosis of congenital dehiscence or osteitis should be made only after thorough microscopic examination, particularly if an ethmoid operation had been performed. The site of injury is usually the roof of the anterior ethmoid, and infrequently the roof of the posterior ethmoid. The roof of the ethmoid is either on the same level as, or higher than, the cribriform plate. For the surgeon, the insertion of the middle turbinate indicates the boundary between the cribriform plate and the roof of the ethmoid (Fig 23). Mesial to the middle turbinate is the cribriform plate with the foramina and lateral to it is the roof of the ethmoid. Manipulations in the superior nasal meatus, i.e., mesial to the middle turbinate, must be performed with great care to avoid injury of the cribriform plate. The danger zone may extend beyond the insertion of the middle turbinate when, occasionally, the most mesial portion of the ethmoid roof, i.e., the area lateral to the middle turbinate, also presents foramina (p. 73). This anomaly renders an ethmoid operation hazardous even though the surgeon keeps strictly lateral to the middle turbinate. The risk increases when the ethmoid roof is low and presents congenital dehiscences, leading to inadvertent removal of the roof and exposure of the dura.

Reports do not always indicate whether the ethmoid roof or cribriform plate was injured, although the prognosis for these injuries differs considerably. If the ethmoid roof is injured the adjacent dura may or may not be injured; if the cribriform plate is injured the dura is always injured. Simple exposure of the dura does not necessarily cause meningitis if the nasal cavity is not packed and chemotherapy is given. The outlook is less favorable when both ethmoid roof and dura are perforated by a blunt instrument and cerebrospinal fluid escapes or when the cribriform plate is injured. Rarely, meningitis does not follow; sometimes it develops several weeks or months later from a common cold and not from the injury, and exceptionally the injury causes an intermittent type of meningitis. However, the usual sequel is fulminating, rapidly fatal meningitis which develops within 24 hours. The value of chemotherapy is not proved, although I saw one case

with recovery after sulfonamide therapy. However, in this case the cerebrospinal fluid showed an increased cell and protein content but no bacteria. Injuries of the cribriform plate during an endonasal sphenoid operation are not common because sphenoid surgery is not frequently performed.

Indirect fractures of the cribriform plate may be caused by



FIG. 59.—Left, section through roof of left nostril. Lm, cribriform plate. CG, crista galli. Ng, superior nasal meatus. N, inflamed mucosa of superior nasal meatus. O, olfactory groove. a, olfactory nerve fibers above cribriform plate. a₁, olfactory nerve fibers below the cribriform plate with perineural infiltration. The sheaths of the nerve fibers are filled with purulent exudate. Right, one olfactory fiber, note leukocyte infiltration in perineural sheath and between nerve fibers. S, corpus arenaceum.

resection of the nasal septum or the middle turbinate. During septum resection a fracture may occur if the surgeon removes the perpendicular plate up to its insertion on the roof of the nose. This is rare. Fractures of the cribriform plate during resection of the middle turbinate are less rare, although the fracture is not always indirect. Frequently it is a direct fracture caused by pushing the conchotome through the cribriform plate. In these

instances meningitis sets in within 24 hours and is fulminating.

Infections of the sheaths of the olfactory bundles (Fig. 39) may occur during resection of the nasal septum and occasionally during resection of the middle turbinate and extraction of polyps. In septum resections which cause a septal abscess or which require forcible packing to arrest a hemorrhage, infection of the nerve sheaths is conceivable. However, in some cases on record the patients died of meningitis from infection of the nerve sheaths although the operation was uneventful. The meningitis does not appear until after the second day, so that chemotherapy may be administered with benefit. After resection of the middle turbinate, an infection of the nerve sheaths should be assumed if the cribriform plate was not fractured and if the meningitis does not develop within the first 24 hours.

Meningitis following extraction of nasal polyps is not uncommon, although not all cases are recorded. The pathologic process is apparently not uniform. When polyps are removed with a conchotome from the roof of the ethmoid or from the superior nasal meatus, a fracture of the cribriform plate is usually the cause of meningitis. When polyps are removed with the cold snare from the middle meatus, a chronic sinus infection may flare up, particularly if the endonasal operation did not achieve satisfactory drainage of the involved sinus. Retention of pus in the sinus in turn causes the chronic sinus infection to extend toward the meninges. The following case may be considered typical.

A man, aged 30, presented himself with polyps filling the right nostril. Between the polyps was creamy pus. The left nostril was normal. The patient seemed acutely ill, complained of severe headache and had fever of 100 F. The polyps were removed by a rhinologist and a large amount of pus escaped from the right nostril. A tampon was not inserted in the nose. The same evening the temperature rose to 102.2 F. and there was projectile vomiting. Sulfonamides were not available at that time. In the next few days the fever did not subside but there were no definite meningeal symptoms and the cerebrospinal fluid was normal. X-ray examination revealed pansinusitis on the right. Two days after extraction of the polyps the right frontal,

ethmoid and sphenoid sinuses were opened. The frontal sinus was small and filled with foul pus. In the ethmoid and sphenoid sinuses were necrotic mucosa and pus, and the anterior part of the lamina papyracea was necrotic. The following day, general improvement was noted, but three days after the operation meningitis was evident, the cerebrospinal fluid was cloudy but contained no bacteria and there was marked leukocytosis. The dura was exposed and appeared normal. Seven days after extraction of the polyps the patient died.

At autopsy no pus was found in the sinuses and the left frontal sinus was small and normal. The dural sinuses were normal. There was extensive meningitis at the convexity and the base of the left frontal lobe and above the left cerebellar hemisphere. Microscopic examination revealed severe purulent inflammation of the mucosa in both superior nasal meati and purulent infiltration of the sheaths of the olfactory bundles on both sides (Fig. 59). There was no fracture of the cribriform plate. The meningitis was due not to injury of the cribriform plate but to infection of the sheaths of the olfactory bundles. In retrospect, the evidence indicates that meningitis was impending before polyp extraction, which simply caused an acute exacerbation of the chronic sinus infection. Owing to the exacerbation, the infection spread rapidly into the nasal mucosa and sheaths of the olfactory bundles on both sides and, ultimately, into the meninges. The crossed spread of infection, consisting of meningitis of the left cerebral hemisphere from an infection of paranasal sinuses on the right, is noteworthy. Other rhinologists have noted such a crossing, but no explanation has been given. In this case sheaths of the olfactory bundles were involved on both sides, and whereas the infection on the right was arrested at the cribriform plate, perhaps owing to the operation, it invaded the meninges on the left side.

Minor endaural manipulations such as extraction of aural polyps and even irrigation of the tympanic cavity or examination with the probe may cause acute exacerbation of chronic otitis and stimulate impending meningitis or sinus thrombosis to a fulminating course. Therefore, all endaural procedures should be omitted when a radical mastoid or labyrinth operation is indicated. Broadly speaking, the same attitude is suggested in the care of paranasal infections. However, in the latter the acute exacerbation is not as hazardous as in the tympanic cavity, unless associated with retention of pus.

Infection may also spread if an endonasal operation is performed in the presence of an acute infection of a paranasal sinus and/or the nasal mucosa, especially when the operation does not aim at better drainage of the involved sinns. For example, in the presence of acute frontal sinusitis, resection of the middle turbinate may relieve the patient by improving the drainage of the involved sinns. In the same case, septum resection alone would not necessarily improve the drainage and fulminating meningitis might follow. It is also hazardous to perform several endonasal operations in rapid succession. All operations may be performed at the same time, but if the second operation must be delayed it should not be scheduled before the tissue, traumatized by the first operation, is adequately healed. Investigations have proved that in the ethmoid, the increased bacterial virulence caused by surgical trauma continues for about 14 days. Some surgeons consider these precautionary measures superfluous, and I am aware that not in all instances of that type is an intracranial complication bound to occur. However, several cases on record demonstrate the hazards of endonasal operations in the presence of acute inflammation of the nose, and since these operations are rarely of an emergency nature there is no reason to take unnecessary risk.

Any micro-organism capable of causing a purulent infection in other parts of the body can also cause purulent meningitis. Both otitic and rhinogenic meningitis are usually caused by a single type of micro-organism, even when it originates in such a source as chronic otitis. Only in meningitis originating in sinus thrombosis, brain abscess or labyrinthitis are several types of bacteria occasionally found in the subarachnoid space. Usually streptococci, staphylococci, pneumococci or influenza bacilli are discovered. Less frequently, *Bacillus coli* or anaerobic bacteria such as *Bacillus pyocyaneus*, *proteus*, *Bacterium haemophilum mucosum*, *Bacteroides fimuliformis* and molds (actinomyces) are the offenders. Anaerobic infections of the meninges occur almost exclusively with chronic otitis. It must be strongly emphasized that infections of both ear and paranasal sinuses may be associated with meningococcic meningitis. Whether the meningococci

enter the meninges through the nasal cavity cannot be definitely stated. In these instances serotherapy is imperative.

Regardless of whether the meningitis is caused by contiguity or continuity, the infection usually involves the base of the brain first. From this initial focus the organisms may be carried farther by the cerebrospinal fluid or blood vessels of the pia, or both. Although there are no fixed routes for the spread of meningitis, certain ones are frequently noticed. Meningitis originating in the posterior cranial fossa remains subtentorially for a comparatively long time, whereas meningitis originating in the middle or anterior cranial fossa, i.e., supratentorially, invades the posterior fossa rapidly. The cause of this variability in time of spread is not known, but it is one reason for the comparatively better prognosis for meningitis which originates in the posterior cranial fossa. From the base of the brain the purulent exudate extends over the convexity of the involved side and, occasionally, of the other side. The exudate usually accumulates over the frontal and temporal lobes, the parietal lobes are less involved and the occipital lobes almost not at all. In some instances of meningitis originating in the nasal cavity or tonsils the convexity of the brain is more involved than the base, owing to infection of the superior longitudinal sinus which spreads toward the convexity. The same distribution is noted in the intermittent type of meningitis of otitic origin (p. 302), although the superior longitudinal sinus is not involved.

The length of the course of fatal meningitis depends on the virulence of the infection. For this reason the autopsy findings differ considerably. In some cases there is only hyperemia of the pia-arachnoid and almost no frank pus at the base of the brain, but the cerebrospinal fluid contains a large amount of cells and protein but no bacteria. In others the entire brain is embedded in pus, although this is not common in otorhinopharyngeal meningitis. A third group may show only an accumulation of pus in the basal cisternae (Fig. 61, p. 284), with the rest of the meninges normal or slightly hyperemic. This condition is often incorrectly called localized meningitis. The findings indicate not localized but diffuse meningitis, the spread of which was arrested by death

in an early stage. In genuine localized meningitis, there must be evidence of a tendency to a walling off of the inflammation by granulations or adhesions.

Microscopically the onset of purulent meningitis is characterized by formation of a purulent exudate around the pial blood vessels. The exudate fills the meshes of the arachnoid and arachnoid cisternae. Hence, at autopsy the pus cannot be wiped off the surface of the brain but can be displaced by pressure on the pia-arachnoid. This observation permits differentiation of purulent meningitis and subdural empyema. At autopsy, when the dura is removed from the brain a large amount of pus will escape if there is subdural empyema, but no pus will escape if there is purulent meningitis because the pus is enclosed in the subarachnoid space.

Even serous infection always involves the brain and choroid plexus. Damage to the brain is due to pressure of the purulent exudate which flattens the cerebral gyri and particularly involves the blood vessels and cerebrospinal fluid. The inflammation usually extends along the pial veins into the brain, causing cortical encephalitis which occasionally increases and forms small cortical abscesses. If the meningitis resolves, the cortical inflammation subsides without leaving scars or other gross changes of the cortex. Some otologists stress the encephalitis and use the term meningoencephalitis instead of meningitis. In my opinion the meningeal inflammation is of greater importance than the encephalitis in the clinical course and outcome, and therefore the term meningitis covers the pathology more satisfactorily. Blanched areas are noted in the cortex either near the site of meningeal infiltration or at a distance from the meninges. These areas are caused by the disappearance of a large number of nerve and glia cells, believed to be due to a disturbance of the local blood circulation.

Since the elaboration, circulation, absorption and chemical constitution of the cerebrospinal fluid are altered by meningitis, the septic fluid is bound to act on the cerebral parenchyma. The exact nature of the changes is not known, but it is certain that the fluid causes mild endependymitis granularis.

The choroid plexus in *meningitis* has not been given proper attention. The plexus usually contains an exudate, although conspicuous infiltration and necrosis are noted only in tuberculous meningitis. Usually an infiltration of the pacchionian bodies interferes with absorption of the cerebrospinal fluid, and often rupture of the pacchionian bodies permits escape of the fluid or purulent exudate into the subdural space.

The meningeal infection may spread through the internal auditory meatus to the inner ear (Fig. 16, p. 39) on both sides, or on one side if the meningitis originated in the temporal bone of the opposite side. This causes serous labyrinthitis which resolves spontaneously if the meningitis is cured, in contrast with the findings in epidemic cerebrospinal meningitis, which also may extend to the inner ear and often causes purulent labyrinthitis and deafness or deaf-mutism.

With control of the meningitis the purulent exudate in the subarachnoid space is replaced by granulation tissue and, ultimately, by connective tissue scars. The scars seldom cause subarachnoid block, i.e., obliteration of the foramina of Magendie and Luschka and/or the cerebellomedullar cisterna, and marked hydrocephalus but may cause moderate hydrocephalus which does not interfere with the patient's mental activity. This opinion is based on clinical experience with children who became deaf-mute after cerebrospinal meningitis and who seldom exhibited either marked hydrocephalus or a mental defect.

PATHOGENESIS AND PATHOLOGY OF SEROUS MENINGITIS

There is great confusion concerning the concept of serous meningitis, attributable largely to the perplexing terminology. Some surgeons consider serous meningitis a clinical entity, using the terms acute hydrocephalus, ntitie hydrocephalus, meningism, generalized eisternal arachnoiditis, collateral meningitis and sympathetic meningitis. Others insist that serous meningitis is not a clinical entity but a preliminary phase of purulent meningitis. Small wonder that several surgeons and neurologists have suggested that the diagnosis of *serous meningitis* be abandoned. The

following discussion is offered strictly from the standpoint of the otolaryngologist, although I am aware that serous meningitis occurs more frequently after injuries of the skull and after systemic infections than after infections of the ear, nose and throat.

A discussion of serous meningitis must be based mainly on clinical experience and results of ventriculography or encephalography. Autopsy observations are seldom available because the patients either recover or die of purulent complications. Nevertheless, I believe that the term serous meningitis should not be abandoned provided the present concept of serous inflammation is applied to the definition of serous meningitis. On page 47 it was emphasized that under normal circumstances fluid filters into the tissue spaces from the arterial portion of the capillary bed, where hydrostatic pressure is higher than osmotic pressure, and is reabsorbed into the venous portion of the capillary bed, where osmotic pressure is higher than hydrostatic pressure. This fluid exchange concerns all osmotically active substances in the blood, except proteins, to which the normal capillary wall is impermeable. Serous inflammation is believed to be due to a change in the permeability of the capillary wall which, owing to the influence of bacterial toxins, becomes permeable to proteins. With this the osmotic pressure of the blood decreases and the back-filtration of fluid from the tissues into the venous portion of the capillary bed is hampered, and fluid containing proteins accumulates between the parenchyma and the capillary bed. The fluid may contain a few lymphocytes if permeability is marked.

Serous inflammation may terminate in (1) spontaneous cure, (2) change into purulent inflammation or (3) organization of the exudate. With spontaneous cure, proteins in the tissues are carried away, probably by the lymphatics, the capillary wall regains normal permeability, osmotic pressure increases and back-filtration into the venous portion of the capillary bed is re-established. There need not be gross changes in the tissues, although there may be microscopic foci of necrosis. In some cases the serous exudate is transformed into connective tissue without passing through a purulent phase and the serous inflammation runs a

chronic course. It is evident, therefore, that serous inflammation is by no means invariably a forerunner of purulent inflammation.

In the cranial cavity serous inflammation of the meninges and brain is often caused by pachymeningitis externa, periphlebitis of the lateral sinus or sinus thrombosis. In the aged, pachymeningitis and sinus thrombosis are rarely associated with serous meningitis, but in children and young people this association is comparatively common. The frequency of serous meningitis from infections of the dural sinuses is based on three factors. (1) The infection of the dural sinuses extends readily into the meningeal and cerebral veins, which empty into the sinus (Fig. 43), causing purulent meningitis or encephalitis or a brain abscess. In some cases, perhaps owing to low grade virulence, only the walls of the venous capillaries become permeable, giving rise to serous inflammation. (2) Sinus thrombosis interferes with the normal flow of blood from the cerebral veins into the sinus, causing increased hydrostatic pressure in the cerebral veins and encouraging the exudation of fluid and protein into the tissues. (3) Granted that the cerebral veins take part in the absorption of cerebrospinal fluid, the increased venous pressure delays its absorption.

The occurrence of serous meningitis without infection of the dura or a dural sinus deserves mention. In several reported cases serous meningitis originated in the tympanic cavity or sphenoid sinus without external pachymeningitis or infection of the dural sinuses. Such cases are not common and there are no autopsy records, so it cannot be stated whether a small focus of pachymeningitis or sinus infection passed unnoticed at operation. There is no anatomic proof of serous meningitis without infection of the dura. From the clinical point of view, this is discussed later (p. 287).

Since the cerebral and meningeal veins freely anastomose, serous inflammation may cause both brain edema and accumulation of fluid in the subarachnoid space, i.e., serous meningo-encephalitis. At the onset the symptoms of inflammation are more conspicuous than those of intracranial hypertension; hence, the condition is called the inflammatory type of acute serous menin-

gitis. This type may be primary or secondary. It is primary when it originates in an infection of the tympanic cavity or paranasal sinuses without intervening pachymeningitis, sinus thrombosis or brain abscess and is secondary when it originates in a brain abscess, localized meningitis or, particularly, sinus thrombosis. Recovery usually follows suitable treatment (p. 297). Otherwise, adhesions form in the network of the arachnoid and between dura and pia-arachnoid. The adhesions give rise to one or multiple encysted collections of fluid in the subarachnoid space which act on the brain like tumors. The fluid is partly cerebrospinal fluid and partly exudate from the blood vessels. This is the hypertensive type of acute serous meningitis. The dura is slightly bulged, and on incision a gush of fluid escapes. The fluid is usually clear, like cerebrospinal fluid, or is slightly hemorrhagic. When the fluid has escaped the cerebral cortex appears to be edematous and the pial vessels are dilated.

This concept implies that the hypertensive phase is always preceded by the inflammatory state, and in many cases the conversion of the inflammatory phase into the hypertensive phase can be noted clinically. In other cases, first observed in the hypertensive phase, an exact history will disclose symptoms of the inflammatory phase at the onset of the illness. In a third group the symptoms of the preceding inflammatory phase are apparently absent, particularly when the hypertensive phase is caused by surgical obliteration of the lateral sinus and ligation of the internal jugular vein. In these cases the symptoms are probably obscured by those of sinus thrombosis. Instances of a hypertensive phase without preceding inflammatory phase and without preceding sinus thrombosis remain problematic, but certainly are not common.

The clinical symptoms suggest that serous meningitis of otorhinogenous origin most frequently involves the convexity of the temporal, parietal or frontal lobes and less frequently the cerebellar hemispheres and base of the brain. This is conceivable if the meningitis originates in an infection of the tympanic cavity or paranasal sinuses. It is less conceivable if the meningitis origi-

ates in thrombophlebitis of the lateral sinus, in which case one would expect the meningitis to be localized in the posterior cranial fossa. Occasionally a collection of fluid is noted between the involved sinus and the cerebellar hemisphere, but more often the meningitis is localized in the region of the temporal and parietal lobes of the involved side. This involvement of the middle cranial fossa in otorhinogenous serous meningitis is in contrast with a similar infection, called generalized cystic arachnoiditis. In this condition the arachnoid is thickened and grayish, owing to increased connective tissue and formation of arachnoid pseudocysts. The arachnoid cisternae, particularly those of the posterior cranial fossa, may be enlarged. Although infections of the ear and paranasal sinuses are frequently mentioned in the etiology of this type of arachnoiditis, I consider them incidental and certainly not common, since generalized cystic arachnoiditis involves especially the posterior fossa and base of the brain, whereas otorhinogenous serous meningitis is usually localized in the middle or anterior fossa.

The inflammatory phase occasionally turns into purulent meningitis, but the hypertensive phase never does. It is usually cured by measures instituted to release the hypertension of the brain and save the function of the optic nerve.

PATHOGENESIS AND PATHOLOGY OF TUBERCULOUS MENINGITIS

Although tuberculosis of the nasal and paranasal mucosa is extremely rare, tuberculosis of the tympanic cavity and inner ear is not uncommon. Diagnosis is often made only by microscopic examination. It usually involves the osseous walls and advances to the dura, causing tuberculous pachymeningitis. In such cases the dura presents tumors which may reach the size of a hazelnut and consist of caseous or fibrocaseous tissue. The tumors grow slowly and cause either no symptoms or symptoms of brain tumor. Usually they do not involve the leptomeninges.

There are two concepts of pathogenesis of tuberculous leptomeningitis: (1) that tuberculous meningitis is caused by dissemination of Koch's bacilli in the blood, and (2) that it is caused

ly a tuberculoma of the pia, choroid plexus, brain or adjacent bone which involves the spinal fluid in the subarachnoid space or ventricles, causing tuberculous meningitis. In tuberculosis of the temporal bone or paranasal sinuses localized tuberculous meningitis may develop first, since the tuberculosis advances very slowly from the temporal bone into the dura, then into the leptomeninges and, finally, into the brain. The localized meningitis may be the source of the generalized form. Such instances are rare, although I have seen two cases.

In one case the tuberculous tumors had penetrated through the tegmen tympani and dura and invaded the occipital lobe. Clinical diagnosis was cerebral apoplexy. In the second case the tympanic cavity was not grossly involved, but tuberculous osteomyelitis of the petrous bone had involved the dura of the posterior cranial fossa, the inner ear on both sides and the cerebellum. Clinical diagnosis was cerebellopontile angle tumor.

In most cases of tuberculosis of the tympanic cavity or ethmoid associated with tuberculous meningitis, both diseases are caused by a hematogenous infection from tuberculous foci elsewhere in the body which involves the tympanic or ethmoid cavity and meninges simultaneously or successively. There is no reason to suppose that tuberculosis of the middle ear or paranasal sinuses is a source of a blood stream infection. This would be possible only if one of the dural sinuses were involved, and tuberculosis of the dural sinuses is so rare that from a practical point of view it does not require consideration.

Occasionally tuberculous meningitis is caused by what seems to be ordinary acute otitis, principally in children. Some otologists even state that in childhood the association of ordinary acute otitis and tuberculous meningitis is more common than that of acute otitis and purulent meningitis. This statement should not be generalized: it depends largely on the incidence of tuberculosis in the particular community. The pathogenesis is not clear. According to one theory, tuberculosis of the tympanic cavity may cause symptoms of acute otitis, and the infection enters the walls of the carotid artery in the carotid canal and thus is car-

ried to the meninges and brain. Other observers suggest that the acute otitis is actually a streptococcus infection and that the tuberculous meningitis does not originate in the ear but is caused by miliary tuberculousis.

As tuberculous meningitis might be associated with what seems to be commnn acute otitis, so might purulent meningitis be associated with definitely tuberculous otitis. The latter condition is not rare, nor is it inconceivable, since in every case of tuberculous otitis there is an additinnal coccus infection which might invade the meninges.

In several cases on record tuberculous meningitis followed surgery for tuberculous mastoiditis, tuberculosis of the nose or tuberculous adenoids or tonsils. Regarding the ear and nose, post-operative tuberculous meningitis occurs only when there is advanced tuberculousis in the body. Therefore the indication for surgery for tuberculous infections of the ear or nose depends entirely on the findings elsewhere: if there is advanced tuberculosis surgery should not be performed. Tuberculousis of tonsils and adenoids is not uncommon, and tuberculous infection of the blood has been noted after operation on infected tonsils and adenoids, but postoperative tuberculous meningitis is extremely rare.

Of least importance is the lymphatic pathway which, exceptionally, carries the infection from the ear to the meninges. A few cases on record indicate that tuberculosis may spread along the sheathis of the facial nerve or the internal auditory meatus into the leptomeninges.

Grossly tuberculous meningitis is characterized by a grayish or greenish exudate filling the subarachnoid space at the base of the brain and extending from the optic chiasma to the pons. From the base the exudate extends especially along the sylvian fissure and central gyrus toward the convexity. Small caseous nodules are seen in the depth of the gyri. The exudate may extend toward the medulla or spinal cord and often involves the sheathis of one or several cranial nerves. It is almost always found in the area of the superior vermis. The choroid plexus is hyperemic and may contain exudate, Koch's bacilli and/or tubercles. The spinal

fluid is clear or slightly cloudy and the amount is increased. The ventricles are more or less dilated, the ependyma of the ventricles may present small nodules and there is definite edema of the brain.

As in purulent meningitis, the cerebral cortex is involved. Lymphoid and plasma cells and leukocytes invade the brain along the pial vessels, or the pial infiltration extends directly, independent of the blood vessels, into the cortex. The infiltrating cells may form nodules which eventually undergo caseation.

SYMPTOMATOLOGY OF PURULENT MENINGITIS

This discussion is concerned with the symptoms of purulent meningitis at the climax of the illness. The symptoms of the initial stage and of the ear and nose are discussed later.

Systemic symptoms.—The patient is acutely ill and shows progressive emaciation. There may be continuous high fever, starting with chills; often the temperature curve shows remissions or intermissions. Before death the rectal temperature may reach 110.0 F. and, rarely, is subnormal. In meningitis due to pneumococcus type III and in aged patients with diabetes the temperature may be only moderately elevated. The pulse rate is not characteristic, in favorable cases depending on the fever and in unfavorable cases showing irregular fluctuations. In the initial stage and after recovery bradycardia may be present. Projectile vomiting is an important symptom. It may or may not be accompanied by nausea, but in either event the patient is not relieved by vomiting. Constipation is usual. The blood shows marked leukocytosis with a more or less pronounced shift to the left, relative lymphopenia and absence of eosinophils and eventually of monocytes. The sedimentation rate is usually high. Fibrinogen and globulin contents of the blood are increased. There is occasionally herpes febrilis.

General brain symptoms.—There are different states of excitement, particularly in adults. There may be restlessness and sleeplessness, and as the condition advances the patient becomes delirious and requires restraint. In other cases there may be

drowsiness and stupor, although the patient promptly responds to needle pricks or similar stimuli, occasionally murmurs unintelligible words, gnashes his teeth and picks at the bed clothes, face, head or genitals. In other words, even in the stuporous state there is always some restlessness, which is usually absent in a stupor caused by a brain abscess. In children restlessness continues for a shorter period than in adults, because children are comparatively soon exhausted. In the initial phase there are peevishness, sullenness and whining. The child does not play and sighs, yawns, moans or hiccups. Later the restlessness increases, there is a hydrocephalic cry and short episodes of stupor and apathy progress to continuous stupor.

Of utmost importance is headache, which varies considerably in localization and intensity. It is always outstanding, even in the initial phase of meningitis. Movements of the head and optic or acoustic stimuli increase the headache. Eyegrounds are and remain normal in most cases, although intracranial pressure may be increased. Optic neuritis occurs occasionally, and papilledema rarely. Perhaps obliteration of the subarachnoid channels and sheaths of the optic nerve in the early phase prevents the cerebrospinal fluid from flowing into the distal part of the nerve.

Cerebrospinal fluid changes are only one symptom of meningitis. For this reason the diagnosis should not be based exclusively on such changes, particularly if repeated examinations are not feasible. The fluid changes must always be related to the clinical findings. Since the pathologic changes at the base of the brain and those in the lumbar portion of the spinal cord are not necessarily the same, the fluid obtained by lumbar puncture does not always offer dependable information concerning the changes at the base of the brain. Nevertheless, certain observations are highly suggestive of purulent meningitis.

The pressure is increased, particularly in the initial phase, and with the patient horizontal may rise to 400 mm. of water. Conspicuously low pressure suggests a block either in the posterior cranial fossa or in the cervical portion of the spinal cord. This diagnosis is confirmed when the cerebrospinal fluid in the man-

ometer does not show oscillations with pulse and respiration and pressure on the jugular veins does not raise the fluid pressure. Marked opisthotonos interferes with the outflow of fluid and may give a false impression of low pressure.

The cerebrospinal fluid is cloudy and occasionally yellowish (xanthochromic). Only in extremely toxic cases of purulent meningitis with death before the formation of frank pus is the fluid clear. Cloudiness is particularly due to the increase of cells. A minimum of 300/3 to 500/3 cells in 1 cu. mm. causes visible cloudiness. In purulent meningitis the number of cells is much greater and may be impossible to count. The number of cells is about the same whether the cerebrospinal fluid is obtained by lumbar or by occipital puncture. If the fluid is collected in several test tubes, that in the first tubes contains more cells than that in the last tubes. If the proportion is reversed, the prognosis for meningitis is believed to be unfavorable. The cells are lymphocytes and polymorphonuclear leukocytes. The predominance of lymphocytes indicates a less fulminating course but does not permit conclusions concerning the final outcome. The lymphocytosis may be temporary. The total protein content is increased to 150-300 mg. and more per 100 cc. Among the various tests, Pandy's yields the most dependable results. In the initial phase the albumin content is greater than the globulin content, later the proportion may be reversed. A high globulin content associated with a small number of cells and eventual discoloration (syndrome of Froin) may be noted if the meningitis causes adhesions in the subarachnoid space — subarachnoid block — with stagnation of the cerebrospinal fluid. With release of stagnation the total protein content may fall, probably accounting for the fluctuations in total protein content in meningitis. The colloid reactions (gold sol, mastic) have no practical importance in diagnosis of purulent meningitis.

The glucose content in both purulent and tuberculous meningitis falls below 40 mg. per 100 cc. In severe cases glucose may disappear entirely. The test for glucose must be performed soon after spinal puncture, otherwise the glucose content decreases or

disappears. If recovery is likely the glucose content may rise; with a new complication, for example, a brain abscess, it falls again. However, the fall in glucose content does not permit definite conclusions concerning the prognosis because in some reported fatal cases of meningitis the decrease was not striking. The reduced glucose content is not related to the number or type of cells or total protein content of the cerebrospinal fluid but is related to the decrease of chlorides and to the glucose content of the blood. Normally the glucose content of the cerebrospinal fluid, particularly in the ventricles, is slightly increased with increase of glucose content of the blood. In meningitis more glucose passes into the cerebrospinal fluid from the blood. Therefore the cerebrospinal fluid content may be misleading if glucose solutions have been administered. The glucose content may also be increased with subarachnoid block.

The chloride content of the cerebrospinal fluid may fall below 600 mg. per 100 cc. in meningitis. The cerebrospinal fluid and blood contain almost the same amount of chlorides, whereas normally the chloride content of the cerebrospinal fluid is greater than that of the blood. The lactic acid content is increased in purulent meningitis, in both cerebrospinal fluid and blood, the increase being more striking in the former. The presence of lactic acid causes a decrease in alkali reserve and lowers the hydrogen ion concentration, the pH being 6.9-7.0 in purulent and 7.5 in tuberculous meningitis. Some authors report an increase of choline content of the cerebrospinal fluid in meningitis.

Bacteriologic examination of the cerebrospinal fluid should be performed soon after the spinal puncture. A culture as well as a microscopic examination of the sediment should be made because occasionally they yield conflicting results. Search for anaerobic bacteria should not be forgotten, particularly in cases of cholesteatoma. Some surgeons hold that diffuse purulent meningitis can be diagnosed only if bacteria are found in the cerebrospinal fluid. This is correct in most cases but should not be generalized because I, like others, have seen cases of otitic and rhinogenous meningitis without bacteria in the cerebrospinal fluid even one or

two days before death. Some statistics indicate that in 25 per cent of cases of otorhinogenous meningitis the cerebrospinal fluid remains free from bacteria. Particularly in rhinogenous meningitis are organisms lacking, or bacteria are seen in one specimen and not on subsequent examinations. In tuberculous meningitis bacilli are found in only 50 per cent of the cases, at most. It is possible that an infection of the tympanic cavity or paranasal sinuses may be associated with idiopathic aseptic meningitis,¹ although I have never seen such a case.

Cisternal instead of spinal puncture has been suggested because the cisterna cerebellomedullaris is nearer the base of the brain. I prefer spinal puncture. In several cases both spinal and occipital punctures were performed, but the latter gave no additional information and usually was more difficult because of neck rigidity. A cisternal puncture is necessary in case of subarachnoid block, which may be located in the region of the foramina of Magendie and Luschka, in the cisterna cerebellomedullaris or somewhere in the spinal cord. In these cases cerebrospinal fluid from different parts of the spinal cord may yield different results. However, subarachnoid block is not common in otitic and rhinogenous meningitis.

Symptoms of hypersensitivity.—Patients with meningitis show vasomotor and pain reactions to stimuli which normally do not reach the threshold of pain sensation. Among the vasomotor reactions, erythema and dermographism are important. In dermographism, red lines appear when the finger-nail is drawn over the skin. A typical symptom of meningitis is hyperesthesia. The patient is hypersensitive to light and noises. Slight pricking, and occasionally even touching, of the skin causes pain. The skin of the abdomen and distinct areas of the skull are particularly sensitive. Pressure on the eyeballs, retrorandibular region and

¹Idiopathic aseptic meningitis presents the clinical symptoms of meningitis, with marked pleocytosis and increase of albumin in the cerebrospinal fluid but normal sugar and chloride contents and no bacteria. Patients do not seem acutely ill. There is fever only at the onset and no, or mild, leukocytosis. The condition invariably subsides in a few days. Persons of any age may be affected. The etiology is unknown, but the cause is not an infection of the ear, nose or throat.

the zygoma and on the fontanel's in infants elicits severe pain.

Motor symptoms and reflexes.—Jacksonian attacks, conjugate deviation of the eyes and twitching of the face muscles are more common in children than in adults. They occur particularly with involvement of the cerebral cortex of the convexity. However, they have also been noted in cases which at autopsy showed the principal accumulation of pus at the base of the brain and only active hyperemia at the convexity. Cortical paralysis of the facial nerve, paralysis of the extremities and motor aphasia occur, but are not common with otorhinogenous meningitis.

Owing to irritation of the posterior roots of the spinal column abnormal reflexes and contractures may develop. The most important is rigidity of the neck. In the incipient phase of meningitis there is stiffness of the neck, and the patient contracts his neck muscles if an effort is made to bend his head forward or backward, although rotating movements of the head to the right and left can be performed. In advanced cases there is fixed opisthotonos and the entire spine becomes almost immobile. Spinal puncture, which is rather difficult in this phase, and removal of a large amount of fluid improves spinal rigidity only temporarily, if at all. Although rigidity of the neck is rarely absent in otorhinogenous meningitis, it is seldom as striking as in meningococcal meningitis, except when there is particular involvement of the posterior cranial fossa. Another contracture involves the abdominal muscles. The surface of the abdomen is deeply sunken under the costal arch and is boat-shaped (scaphoid abdomen). This is not very common with purulent meningitis. Particularly in children, contracture of the legs is frequently noted. The legs are flexed and pulled toward the chest. However, these contractures can be released, despite considerable resistance.

Irritation of the posterior roots of the lumbosacral spine also increases the muscular reflexes, as indicated by the signs of Kernig, Lasègue and Brudzinski. To test for Kernig's sign, with the patient lying on his back the thigh is slowly or abruptly flexed on the trunk to a right angle. A reflex contracture of the hamstring muscles, not necessarily painful, causes resistance to this pro-

cedure. The same contracture is caused by raising the patient's trunk to form a right angle with the thighs. To test for Lasègne's sign, the patient's foot is grasped with one hand, the other hand is placed on the knee and, keeping the leg stiffly extended, the thigh is flexed on the pelvis, causing a severe pain in the hips or higher up in the course of the sciatic nerve. The test for the neck sign of Brudzinski consists in partial flexion of the hip and knee joints when the head is flexed passively on the chest. The signs of Kernig and Lasègne are not dependable if a spinal puncture was performed a short time before.

The tendon reflexes are often increased in the initial phase of meningitis and diminished in the advanced phase. Babinski's reflex² and ankle clonus are present in some cases and absent in others. So also are the following: Edelmann's reflex, consisting of dorsal flexion of the great toe when the leg is bent at the hip joint while the knee joint is extended; Gordon's reflex (dorsal flexion of the great toe caused by pressure on the calf muscles); Oppenheim's sign (dorsal flexion of the great toe caused by stroking or scratching of the median or inner surface of the leg); Ghaddock's sign (dorsal flexion of the great toe caused by stroking of the skin over the external malleolus); Bind's shoulder sign (movement of the shoulder up and forward on forcible turning of head to the opposite side); Magnus-de Kleyn's sign (raising of the leg and arm on forcible turning of head to the opposite side). Some surgeons claim that the absence of Babinski's sign suggests a favorable prognosis. After spinal puncture the tendon reflexes may disappear. In the terminal phase the sphincters of the rectum and urethra become paralyzed.

Symptoms of the cranial nerves.—In advanced cases the pupils change frequently in size, one pupil being larger than the other. In the terminal phase the pupils become enlarged and do not react to light. There is occasionally paralysis of the abducens nerve on the involved side, sometimes on the other side. This, however, is more common in localized and serous meningitis than in diffuse purulent meningitis. Vertical nystagmus indicates a

²Babinski's reflex is usually noted in normal infants up to 12-14 months

poor prognosis in both meningitis and brain abscess. In one case of meningitis with vertical nystagmus Grabseheid discovered an extensive, almost symmetrical, deposit of lipoids in the cells of Deiters' nucleus on both sides. Symptoms of trigeminal nerve involvement are infrequent.

All of these symptoms indicate meningitis, but they do not permit definite conclusions concerning its localization, i.e., whether there is an accumulation of pus at the base or at the convexity of the brain.

PROGNOSIS FOR PURULENT MENINGITIS

There are few diseases in otolaryngologic practice for which the prognosis was so decisively improved by the introduction of sulfonamides and penicillin as purulent meningitis. Some surgeons claim that since introduction of the sulfonamides the mortality rate has fallen from nearly 100 per cent (more exactly, 60-70 per cent) to between 20 and 45 per cent. In my opinion these figures challenge some critical remarks. A review of the recent literature indicates that whereas the cases with recovery after chemotherapy were reported at length, little mention was made of cases fatal despite chemotherapy. Furthermore, the authors usually dealt with "meningitis," not considering that this is a collective term which includes various types, i.e., types with a good prognosis from the onset and types for which the prognosis is extremely unfavorable. For the same reason, one cannot speak of the prognosis for laryngeal carcinoma in general terms, since the prognosis for a carcinoma of the vocal cords differs considerably from that for carcinoma involving the piriform sinus.

To evaluate the efficiency of chemotherapy the principal factors which influence the prognosis for meningitis must be considered.

General physical condition.—This depends on the patient's age. In elderly patients invariably the prognosis must be guarded, especially when the cardiovascular system is involved, regardless of whether chemotherapy is used or not. In young persons, particularly children, the prognosis is more favorable, as shown

in Figure 60. Eighty-three cases of meningitis from acute otitis were reviewed. In all cases micro-organisms, particularly streptococci and pneumococcus type III, were found in the cerebrospinal fluid. Forty-nine patients were treated with sulfonamides and 34 were not. Both curves present a peak in the age group

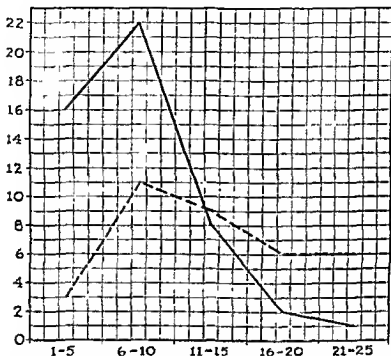


FIG. 60.—Solid line indicates cases of meningitis following acute otitis, cured without sulfonamide therapy. Broken line indicates those treated and cured with sulfonamides. Abscissa indicates patient's ages, ordinate shows number of patients.

6-10 years; in other words, most frequently a cure was achieved in patients between 6 and 10 years regardless of whether chemotherapy was used or not. Comparison of the cases treated with and without sulfonamides suggests that there is no difference in the results. This conclusion, however, would not be correct. The cures achieved by administration of sulfonamides were reported during two years, whereas those achieved without sulfonamide therapy were reported over 39 years. This definitely proves the

efficiency of the drugs. Furthermore, after the tenth year of life the sulfonamide curve shows a gradual descent, whereas the other curve falls rapidly, indicating that the sulfonamides achieved a considerable number of cures in patients over 10 years who could not have been saved without sulfonamide therapy. However, both curves stop at the age group 21-25 years, indicating that after the twenty-fifth year the sulfonamides do not materially improve the prognosis for meningitis due to acute otitis. Perhaps penicillin will be more successful in these instances.

Strain and virulence of bacteria.—Certain strains of streptococci do not respond to sulfonamides, and certain micro-organisms such as *Bacillus coli* and *proteus* are in all circumstances resistant to sulfonamides. Often sulfonamide therapy fails in infections due to pneumococcus type III, not only because of a distinct resistance of the micro-organisms but also because administration of the drug is frequently delayed. In these cases the meningitis may have a long latent period, until suddenly fulminating meningitis appears. In some of these cases cure could probably be achieved were the sulfonamides administered during the latent period.

Involvement of the brain.—The ultimate cause of death in meningitis is not known. In some cases pneumonia causes death; in other rare instances a blood stream infection may be responsible. When death is due neither to pneumonia nor to septicemia, the cause must probably be sought in the brain. However, the marginal encephalitis (p. 260) associated with meningitis neither causes definite neurologic symptoms nor threatens the patient's life. Of greater importance may be the brain edema. The theory has been advanced that the increase of lactic acid causes a genuine acidosis of the cerebrospinal fluid which is not compensated and which will kill the patient in a short time when it involves the blood. The acidosis and also the reduction of the alkali reserve of the cerebrospinal fluid act on the brain and cause both local and general brain symptoms. Death is believed to ensue from local acidosis of the brain if the acid-alkali balance

cannot be restored. In agreement with this theory is the observation that the lowering of the alkali reserve of the cerebrospinal fluid points to an unfavorable prognosis. Also, cases of meningitis presenting a multitude of symptoms are to be considered more serious than those showing only a few neurologic symptoms.

Pathogenesis of meningitis.—If surgery is performed at the proper time, all cases of purulent meningitis which pass through a phase of localized meningitis have a more favorable outlook than those which are more or less diffuse from the onset. In cases of the first type, if an operation is performed while the meningitis is still localized, cure often follows even without chemotherapy. However, if surgery is delayed until the localized meningitis has passed into the diffuse stage, the outlook is poorer than in cases which are diffuse from the onset.

TREATMENT OF PURULENT MENINGITIS

Several decades ago Jansen stated that the first symptom of meningitis is the last summons to operate. I adhere strictly to this dictum. However, Jansen's statement does not answer the question of what to do when the first symptoms pass unrecognized and the patient first comes under observation presenting full-blown meningitis. Formerly an operation was performed on the temporal bones or paranasal sinuses, but experience has taught that the operation is of no benefit and often seems to hasten death. For this reason, in cases of otitic or rhinogenous meningitis which have passed the initial phase it is suggested that conservative treatment be tried first. If the patient responds favorably, the operation should be performed later.

Conservative measures include antiseptic and supportive treatment and drainage of the subarachnoid space. The administration of sulfonamides is discussed on page 163. It is essentially the same for meningitis as for septicemia. Some surgeons advise intrathecal injection of the sulfonamides. However, 15 minutes after intrathecal injection large amounts of the drug are found in the blood. Thus, within the subarachnoid space, absorption prevails over diffusion, and the drug acts only on the part of the

spinal cord where the injection was given, the action not extending to the brain. Furthermore, if, *in vitro*, a sulfonamide is added to the cerebrospinal fluid in an amount equal to that found in the fluid after intrathecal injection, an increased bacteriostatic effect cannot be detected. Therefore in both meningitis and septicemia oral or intravenous administration of the sulfonamides is the method of choice.

Although the sulfonamides (except sulfathiazole) pass through the blood-brain barrier, penicillin does not penetrate the subarachnoid space in appreciable amounts following intravenous or intramuscular administration. In normal subjects, 10,000-20,000 units of penicillin in 10 cc. of isotonic saline solution given intrathecally is slowly absorbed and slowly excreted in the urine. In patients with meningitis both processes appear to be accelerated, although not as much as with sulfonamides. Toxic signs, including headache, vomiting, increased intracranial pressure and pleocytosis of the spinal fluid, have occasionally followed the injection of 10,000 units into the subarachnoid space. Nevertheless, in meningitis a minimum of 10,000 units in physiologic saline, in concentration of 1,000 units per cc., should be injected once or twice daily. Subsequently, 5,000-10,000 units per dose may be injected at intervals of 20-24 hours. Intramuscular injections up to 200,000 units daily or intravenous injections should be given at the same time if necessary. Treatment is continued for two or three days or longer, as needed.

Penicillin and the sulfonamides can be given simultaneously, particularly in certain mixed infections, since apparently the two drugs are synergistic in therapeutic action. Determination of proper dosage may be difficult, for hard and fast rules do not exist. All depends on the seriousness of the case and the surgeon's experience. Penicillin prophylaxis should be employed in all cases of otitis and sinus infections presenting vague symptoms of meningeal involvement before operation. Intramuscular injection of 10,000-120,000 units every four hours for two or three days will suffice.

The injection of antiseptic gas (acetylene) into the subarach-

noid space did not prove successful. The use of serum and vaccines was replaced by modern chemotherapy. Only in pneumococcic infections, a type-specific antipneumococcus serum may be valuable, since neither penicillin nor the sulfonamides act on pneumococci with uniform effectiveness. Sufficient antipneumococcus type-specific rabbit serum should be given to establish an excess of antibodies in the circulation. To make certain, a 1:1,000 dilution of the type-specific polysaccharide in saline is injected intracutaneously. A wheal develops at the site of the injection within 20 minutes if there is an adequate amount of circulating antibodies.

It must be emphasized that chemotherapy acts primarily on the meningeal infection; its effectiveness on the infection in the temporal bone or paranasal sinuses is less certain. Therefore, in a given case of meningitis, if cerebrospinal fluid findings improve, the final outcome still depends on the infection in the temporal bone or paranasal sinuses. If the infection tends to spontaneous cure or if it responds to chemotherapy, definite cure can reasonably be expected. However, if the infection in the temporal bone or paranasal sinuses remains active, improvement of the meningitis may be only temporary. This occurs particularly in adults or old persons. For this reason, chemotherapy has rendered the task of the surgeon more difficult. Formerly, in all such cases an operation was performed on the temporal bone or paranasal sinuses, but now the surgeon must decide whether the infection in the temporal bone or paranasal sinuses is definitely arrested or has passed into a temporary phase of latency. If this is impossible, it is safer to operate and then continue with chemotherapy.

As in septicemia, supportive treatment is essential, the purpose being to increase the fighting forces of the body and to avoid dehydration and brain edema. The measures required to maintain resistance of the body and to combat dehydration and brain edema are discussed on page 175.

In the past, and occasionally now, drainage of the subarachnoid space played a considerable part in the treatment of meningitis. Various methods were employed, such as incision of the

dura, frequent spinal punctures and occipital and ventricular punctures, but no convincing results were obtained. Drainage of the subarachnoid space seemed logical (1) to remove septic fluid from the brain and (2) to reduce pressure in the skull. This reasoning seems incorrect. Meningitis is, primarily, not a disease of the spinal fluid but a disease of the leptomeninges and eventually of the brain. Furthermore, the cerebrospinal fluid is not an inflammatory exudate; rather, it dilutes the inflammatory exudate and, although not possessing marked bactericidal properties, is not a good culture medium for bacteria. For this reason, the removal of cerebrospinal fluid in meningitis cannot be compared with the evacuation of pus from a cavity in the body.

A procedure based on an interesting theoretical concept is "forced drainage of the central nervous system." This consists of administration of water by mouth or intravenous or subcutaneous injection of hypotonic saline solution while allowing the free escape of cerebrospinal fluid. The purpose is physiologic lavage of the central nervous system and a partial washing-out of inflammatory products from the depths of the tissues to the surface. Despite the ingenious concept, the method did not become popular because of the danger of brain edema and discomfort to the patient. Moreover, a large amount of pus in the cerebrospinal fluid may block the needle and render the drainage impossible.

Regarding reduction of pressure, it may be granted that pressure in the skull is decreased by spinal puncture and that the relationship between cerebrospinal fluid and blood pressures will be normalized, although there is no actual proof. However, the primary concern in medical care of meningitis is not the pressure of the cerebrospinal fluid but the increased pressure due to the brain edema. There is no evidence that drainage of the subarachnoid space is more effective than hypertonic solutions against brain edema. Incisions of the dura have not proved more successful than repeated spinal punctures. The incisions are closed rapidly either by fibrinous exudate or by the formation of a small brain hernia at the site of incision. Incision of the lateral pontile cistern is frequently followed by marked escape of fluid for several

days provided the labyrinth is surgically removed. If this has not been done, this type of drainage of the subarachnoid space is seldom efficient. Drainage of the subarachnoid space was abandoned by some surgeons who claim that it is definitely harmful by encouraging the formation of adhesions in the subarachnoid space.

I believe, theoretically, that free circulation of the cerebrospinal fluid may be altered by draining the subarachnoid space. For this reason, fluid which is withdrawn should be replaced by air. The technic is discussed on page 65. In meningitis, 10-30 cc. of cerebrospinal fluid is withdrawn with the patient in sitting position. If the pressure is high the fluid is withdrawn very slowly, with control of pulse and breathing. Air is then injected in an amount about 5 cc. less than that of cerebrospinal fluid withdrawn. The purposes of this treatment are: (1) to make the patient more comfortable temporarily, i.e., to relieve the intense headache; (2) by air inflation to cause a slight leukocytosis of the fluid, which may increase the phagocytosis of bacteria, and (3) to prevent eventual formation of adhesions in the subarachnoid space. The procedure may be repeated.

OTIC MENINGITIS

The primary type of otitic meningitis may originate in a purulent infection of the tympanomastoid cavity, labyrinth, pneumatic cells of the petrous bone or the pneumatic cells of the malar process. The secondary type originates in a sinus thrombosis, internal pachymeningitis or brain abscess. The low incidence of otitic meningitis originating in osteomyelitis of the temporal squama makes it of minor importance.

The secondary type is more common than the primary. Not infrequently meningitis clinically considered to be primary is actually secondary. The following case is an instructive example and demonstrates the difficulty in classifying otorhinogenous meningitis.

A man, aged 63, had pain and diminution of hearing on the right. The next day scanty discharge from the right ear was noted and pain and fever persisted. A paracentesis was performed. The symptoms

subsided and the patient became ill with what was considered to be grip. There was again discharge from the right ear and the temperature rose to 102.2 F. daily. There was no headache, earache or symptoms of mastoiditis. Since the temperature did not subside and the discharge increased, he was hospitalized a month after onset of the ear infection. Dizziness, pain and chills were not present, but the superior posterior wall of the external auditory canal on the right sagged and the temperature was 102.4 F. There were slight jaundice of the skin and sclerae and pallor of the optic disks due to arteriosclerosis. A simple mastoid operation revealed all cells filled with pus and granulations. The sinus wall was normal, the dura of the middle cranial fossa was covered by a fine layer of fibrin and the cerebrospinal fluid did not contain bacteria and pressure was normal. In the next 10 days the temperature fluctuated between 97.3 F. in the morning and 103.2 F. in the evening. There were no chills or meningeal symptoms, but a large amount of pus escaped from the superior and posterior angles of the bony cavity. Babinski's and Oppenheim's signs were present on the right. Sulfonamides were not yet available.

The wound was re-opened, and purulent exudate was discovered in a pneumatic cell in the petrous angle, but the dura and sinus were normal. The sinus was injured close to the jugular bulb and blood escaped from the sinus. The cerebrospinal fluid was hemorrhagic, did not contain bacteria and was under low pressure. After the operation meningitis appeared and 10 days later the patient died. Autopsy revealed diffuse purulent meningitis which was considered primary, i.e., to have originated in the mastoiditis. However, microscopic examination of the temporal bones proved that the meningitis was secondary to long-standing purulent thrombophlebitis of the jugular bulb which was not recognized during life.

There are three types of otitic meningitis: localized, serous and acute purulent.

LOCALIZED MENINGITIS

Localized meningitis is a purulent inflammation of the leptomeninges which is localized and remains so for a long, indeterminate period of time.

Pathology.—Localized meningitis occurs in (1) all infections which travel by contiguity, (2) all types of meningitis which originate in the posterior cranial fossa and (3) brain abscess, sinus thrombosis and internal pachymeningitis.

Among the infections which travel by contiguity, cholesteatoma of the tympanic cavity not infrequently causes localized meningitis as long as there is no acute infection of the cholesteatoma. Among the types of meningitis which originate in the posterior fossa, infection of the arachnoid cisternae near the petrous apex, i.e., the pontile cisterna and the cisterna of the gasserian ganglion (Figs. 61 and 62), runs a peculiar course

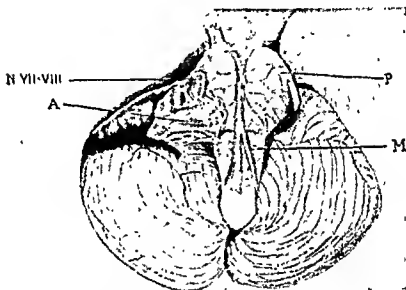


FIG. 61—Localized infection of cisterna pontis (A). N VII-VIII, facial and acoustic nerves. P, pons. M, medulla oblongata. (After Alexander.)

(p. 256) The subarachnoid spaces over the cerebral and cerebellar hemispheres contain small cavities which soon become obliterated by connective tissue, whereas the arachnoid cisternae at the base of the brain are large cavities which may hold large quantities of purulent exudate. Therefore meningitis localized over the cerebral or cerebellar hemispheres may eventually undergo spontaneous cure, as in brain abscess and, occasionally, in sinus thrombosis. Dura, arachnoid and pia are replaced by a thick membrane consisting of firm connective tissue which may contain foci of active inflammation. The membrane is firmly adherent to the cerebral or cerebellar cortex (Fig. 63). In an arachnoid

cisterna, however, the amount of accumulated pus is too great to be replaced by connective tissue, and eisternal meningitis invariably causes diffuse meningitis if surgical drainage is ineffective. A localized infection of the pontile eistern is rarely caused by purulent labyrinthitis or petrositis.

Symptomatology.—Localized meningitis is not a clinical entity.

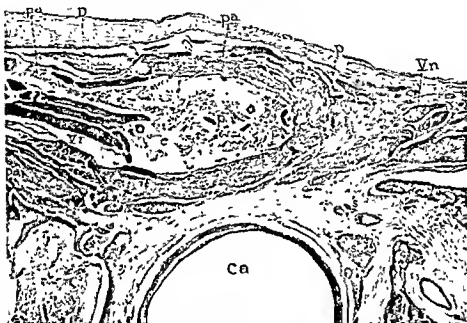


FIG. 82.—Localized meningitis in the gasserian ganglion cistern (*c*). *Ca*, internal carotid artery; *s*, branches of sympathetic nerve, *g*, gasserian ganglion, *p*, purulent exudate in cistern and between divisions of trigeminal nerve (*Vn*), *Vr*, roots of trigeminal nerve, *pa*, pia-arachnoid sheath of trigeminal nerve, *D*, dura mater.

The differential diagnosis of localized meningitis, internal pachymeningitis and brain abscess is difficult and often impossible, since localized meningitis does not cause definite symptoms. For this reason, the diagnosis of localized meningitis is nearly always tentative and based on the presumable pathology of the case. For example, if a patient with cholesteatoma presents slight meningeal symptoms, localized meningitis should be considered. On the other hand, if a youngster with acute otitis presents the same meningeal symptoms, an initial phase of diffuse, not local-

ized, meningitis should be considered. The results of spinal puncture do not permit definite conclusions. Some surgeons omit spinal puncture in cases of eventual localized meningitis to avoid spread of the meningitis. However, the hazards of spinal puncture in localized meningitis are not great provided too large an amount of fluid is not removed rapidly.

Meningitis localized in the pontile cistern causes essentially



FIG. 63—Localized meningitis on posterior surface of left cerebellar hemisphere (arrows). There was a small cerebellar abscess on the anterior edge of the hemisphere. Note edema of the hemisphere. (After Grabscheid.)

the same symptoms as petrositis. Occasionally the predominance of cerebellar symptoms is responsible for the incorrect diagnosis of cerebellar abscess. Cisternal meningitis may run a protracted course over several weeks, during which the meningeal symptoms disappear and re-appear. Even this is not absolutely indicative of cisternal meningitis, since it may also be noted in the intermittent type of purulent meningitis and in serous meningitis.

Treatment—Localized meningitis caused by a brain abscess or sinus thrombosis does not require special care. That caused by an infection of the temporal bone requires removal of the focus of infection by appropriate surgical procedures and post-operative chemotherapy.

ACUTE SEROUS MENINGITIS

Serous meningitis is an accumulation in the subdural spaces of the brain of cerebrospinal fluid and a serous exudate, rich in albumin, caused by abnormal permeability of the venous portion of the capillary bed. There is an inflammatory and a hypertensive type.

Inflammatory type.—**PATHOLOGY.**—This type occurs in children and young persons, and seldom in adults past 30. Formerly, acute serous meningitis was most often seen with cholesteatoma involving the inner ear. Now such cases are considered to be instances of incipient purulent meningitis because they almost invariably become purulent unless an operation is performed immediately and chemotherapy administered.

True serous meningitis may be primary, secondary or post-operative. The primary type is caused especially by acute otitis, usually appearing in the first week of the otitis and occasionally even before rupture of the drum membrane. It is probably caused by an infection which travels along the anastomosing blood vessels. Chronic otitis seldom causes serous meningitis, except in children. The secondary type is most frequently caused by infections of the lateral sinus, periphlebitis or thrombophlebitis.

The postoperative type usually becomes manifest after a simple mastoid operation. Failure to operate at the proper time and failure to eliminate the entire infection in the temporal bone are usually responsible for the postoperative development of serous meningitis. A common error is the performing of a simple mastoid operation for recurrent acute otitis. This type of otitis becomes evident when an acute infection involves a tympanic cavity containing adhesions caused by previous attacks of acute or chronic otitis. These cases are often not properly evaluated, although there is usually evidence suggesting recurrent acute otitis. Such evidence includes: a history of several attacks of acute otitis, but no discharge in the intervals; the finding of a poorly or nonpneumatized mastoid process in apparently acute otitis, and discharge from the tympanic cavity without preceding

pain. Localized abscesses have a tendency to form in the tympanic cavity in these cases, so that simple mastoid operation rarely accomplishes proper drainage of the exudate in the tympanic cavity. In fact, this operation may cause a flare-up of the tympanic infection which, in turn, may cause serous meningitis a few weeks later. This complication can be avoided if, in recurrent acute otitis associated with mastoiditis, the operation is extended toward the epitympanum. Some cases require a modified or even complete radical mastoid operation.

Another cause of postoperative serous meningitis is failure to establish the correct diagnosis and to perform the required surgery in cases of sinus thrombosis. There is a considerable number of such failures on record. These cases usually have the following history: although there was clinical evidence of sinus infection, a simple mastoid operation was performed without exploring, occasionally even without exposing, the lateral sinus, and serous meningitis followed in a few weeks. The meningitis may resolve if the sinus thrombosis undergoes spontaneous cure, or may become purulent if the thrombosis is progressive.

In a third group, serous meningitis may follow surgery for mastoidism (p. 188). Fever, increased lumbar pressure and eventually cloudy cerebrospinal fluid may appear after the operation, particularly in older children with a well pneumatized mastoid process. The serous meningitis gradually disappears, and further surgery on the temporal bone, such as exposure of the dura or sinus, is seldom necessary, although such surgery is frequently performed. Chemotherapy will probably shorten the course in these cases. Serous meningitis may also occur if, during a simple mastoid operation on an infant, the dura in the antrum is exposed and the retroauricular incision is primarily closed. In these cases, granulations form in the antrum adjacent to the dura, and if the tympanic cavity is reinfected, serous meningitis may result. For this reason, exposure of the dura in infants is not as harmless as it is in adults. Serous meningitis may ultimately follow a radical mastoid operation, particularly if it results in atresia of the external auditory canal.

SYMPTOMATOLOGY.—The inflammatory type of acute serous meningitis is characterized by symptoms of meningeal irritation. The patient is restless and sleep is disturbed, although the restlessness is not as striking or as progressive as in purulent meningitis. Apathy and stupor are less common and point rather to purulent meningitis, particularly in infants. Progressive emaciation, continuous vomiting and delirium are also more suggestive of purulent than of serous meningitis. Occasionally the patient complains of vertigo. Headache, particularly frontal headache, is constant, and so is fever. The fever is not characteristic, but it may rise to 104 and 105 F. especially at the onset. In serous meningitis due to sinus thrombosis the fever shows remissions suggestive of sinus thrombosis. Despite the high temperature the patient's condition does not deteriorate as rapidly as in purulent meningitis. Chills are infrequent except in serous meningitis due to sinus thrombosis. The pulse rate follows the temperature fluctuations and occasionally is slow. The blood shows leukocytosis but no marked shift to the left. There are no bacteria in the blood unless there is associated sinus thrombosis. The eyegrounds are normal or there is low grade papilledema rising to about 2 D. and associated with hemorrhages in the retina. Lumbar puncture may reveal normal pressure, but more often the pressure is raised to 250-350 mm. of water. The fluid is clear or slightly cloudy and shows a moderate increase of lymphocytes. The protein content is increased even though the fluid is clear. Bacteria are invariably absent. Sugar and chloride contents are normal. The ventricles are of normal or subnormal size; if there is internal hydrocephalus, it is probably not caused by otorhinogenous serous meningitis.

A boy, aged 15, with recurrent otitis and mastoiditis on the right, underwent a simple mastoid operation, and about three weeks later the first symptoms of serous meningitis appeared. Frequent lumbar encephalographies revealed normal ventricles, but the subarachnoid spaces over the right cerebral hemisphere and the cisterna pontis on the right were not filled with air (Fig. 64). When a lateral view of the skull was taken with the right side of the skull uppermost, a filling of the subarachnoid spaces on the right was achieved. These observations indicated right-sided brain edema. The diagnosis was

corroborated by the neurological examination, which revealed jacksonian attacks and hemiparesis on the left, absence of the left triceps and radial reflexes and abducens paralysis on the right. The patient was cured by frequent lumbar punctures.

Symptoms of hypersensitivity (pp. 272 f.) are present, particu-



FIG. 61.—Serous meningitis after recurrent acute otitis, acute mastoiditis and peripneumonia of sigmoid sinus on the right. Encephalogram after withdrawal of 30 cc. of cerebrospinal fluid and inflation with 25 cc. of air, anteroposterior view. Arrows indicate subarachnoid spaces on the left filled with air. A, cisterna pontis on the left filled with air. The same spaces on the right are not filled with air.

larly in children. The occurrence of motor symptoms and changes of reflexes (p. 273) depend on the extent of brain edema. Spinal cord symptoms, such as pain in the spine, buttocks and thighs and paralysis of the rectum and urinary bladder indicate purulent rather than serous meningitis. Of the cranial nerves, the abducens is most frequently involved, usually on the affected side, although

at times on the opposite side. The oculomotor and facial nerves are involved in a few instances.

DIFFERENTIAL DIAGNOSIS.—This is difficult as far as purulent meningitis is concerned. Formerly, the differential diagnosis was based mainly on cerebrospinal fluid changes, but there is no uniformity of opinion regarding evaluation of the fluid. Some surgeons consider increased lumbar pressure and normal fluid to be suggestive of serous meningitis, while others emphasize an increase of protein and cell contents. Each finding must be considered compatible with the diagnosis of both serous meningitis and an incipient phase of purulent meningitis. Likewise, final recovery cannot be considered a significant feature of serous meningitis, since purulent meningitis is no longer invariably fatal.

Apparently the differential diagnosis cannot be based on a single symptom. The following observations are suggestive but not decisive in this respect. (1) The patient's age. Broadly speaking, serous meningitis must always be considered when meningeal symptoms occur in children and young persons. In adults over 30, serous meningitis is not common. (2) Type of otitis. If in an early stage of acute otitis, even before rupture of the drum membrane, meningeal symptoms such as restlessness, fever and headache occur in a child or young person, the diagnosis of serous meningitis must be assumed, except during epidemics of influenza, when acute infections of the tympanic cavity may lead to purulent meningitis with unusual rapidity. Serous meningitis must also be considered in children or young persons when meningeal symptoms appear a few weeks after a simple mastoid operation. (3) General condition of the patient. Purulent meningitis rapidly breaks down resistance, particularly in children, resulting in rapidly and rapidly progressing emaciation. The child with serous meningitis may be quite active; when he falls asleep it is a natural reaction of fatigue, not a sign of systemic deterioration by toxins. This observation is of utmost importance. (4) Cerebrospinal fluid. Although lumbar pressure and the cell and protein contents are not dependable criteria, progressive reduction of sugar content and the appearance of bacteria point to

a purulent meningitis. (5) Final recovery. Serous meningitis is not fatal. If a patient dies of meningitis, it is purulent, even though the cerebrospinal fluid was clear. The concept of "malignant serous meningitis" is not in agreement with the modern concept of serous inflammation.

The differential diagnosis of serous meningitis and localized purulent meningitis is even more difficult. One should remember that localized purulent meningitis may occur in cholesteatoma of the tympanic cavity, a condition which rarely causes serous meningitis.

Prognosis.—For both primary and secondary serous meningitis the prognosis is good if the original infection is properly managed. Otherwise prognosis must be guarded. Resolution occurs if the underlying otitis or sinus thrombosis undergoes spontaneous cure. In such cases the meningitis may continue for several weeks or may display intermissions and recurrences until ultimate recovery. The following case is instructive.

A girl, aged 10, with a positive Pirquet reaction, had acute otitis on the right of two months' duration. On admission the temperature fluctuated between 100.4 and 101 F. A simple mastoid operation revealed a well pneumatized mastoid process; the exposed dura and sinuses were normal. Postoperatively, the fever decreased by lysis. Sulfonamides were not given. Twelve days after the operation there were high fever, edema of the right upper lid and 13,700 leukocytes in the blood, with a slight shift to the left, but there was no papilledema. The mastoid incision was enlarged, and granulations were removed from the perilabyrinthine and retrosinus cells. There was again gradual recovery, but one month after the first operation the temperature rose to 102.2 F., with vomiting, shivering and facial pallor, but no papilledema. Lumbar pressure was increased and the fluid cloudy. A radical mastoid operation revealed the tympanic cavity filled with granulations, the dura normal and the sinuses covered by fibrin and granulations, but no thrombus. The superior angle of the petrous bone was removed. Four days later lumbar pressure was normal and the fluid clear. Six weeks after the first operation there was again vomiting, but no fever. Three weeks later the temperature was 99 F. There were frontal headache on the right, vomiting, slight rigidity of the neck and Oppenheim's sign on the left. Kernig's and Babinski's signs were not present. Lumbar pressure was slightly increased and the fluid was

yellowish and cloudy and contained 800/3 cells; the Nonne-Apelt and Pandy reactions were 2 plus. Air inflation of the spinal canal led to disappearance of all symptoms the following day. There was no recurrence of meningeal symptoms and the child remained well.

Not all cases have as favorable a course as this one. In some the serous meningitis continues for a few weeks without alarming symptoms. Then suddenly, without warning, marked meningeal impairment develops. The patient (usually a child) becomes semicomatose and pale, respiration is labored and the pulse weak or very slow; there may be deviation of the eyes, a sluggish reaction of the pupils, ataxia, ankle clonus, a positive Kernig sign and diminished abdominal reflexes. This acute breakdown may be the forerunner of the hypertensive state or of purulent meningitis. The change from serous to purulent meningitis is likely if the mastoid or lateral sinus infection is progressive. In certain epidemics, as in the influenza epidemics in Vienna in 1928, this change occurred frequently in the early phase of acute otitis. Meningeal symptoms appeared almost simultaneously with the acute otitis; the spinal fluid was cloudy the third or fourth day of the otitis and contained bacteria one to two days later. The prognosis was unfavorable. One must assume that the offending organisms were especially virulent. Chemotherapy was not available.

If the inflammatory type of serous meningitis advances to the hypertensive type, the symptoms of the ear infection and of meningeal irritation gradually disappear and are replaced by those of intracranial hypertension.

TREATMENT.—In primary serous meningitis a paracentesis of the drum membrane is occasionally all that is necessary to cure the infections of both ear and meninges. In acute otitis with surgical mastoiditis a simple mastoid operation should be performed, and in recurrent otitis the conservative radical and the complete radical operations are the methods of choice. The dura should be exposed. If a pathologic process is discovered in the mastoid extending to one of the cranial fossae, the dura is exposed at this site. If no pathologic process is found the dura of both cranial fossae should be exposed.

Secondary serous meningitis caused by sinus thrombosis requires the sinus operation; ligation of the jugular vein should be omitted unless there is advanced septicemia. If the operation is performed at the proper time, the meningeal symptoms rapidly disappear, regardless of whether chemotherapy is given or not. A special problem arises in cases of meningitis in the first week of acute otitis. Although there is no surgical mastoiditis and although the principal infection is in the tympanic cavity, not in the mastoid process, a simple mastoid operation is often performed unnecessarily (p. 188). In these cases chemotherapy is preferable. If penicillin is prescribed, pyrimidon (3-5 Gm. daily) should be added, to constrict the blood vessels and reduce the exudation of serous fluid.

Hypertensive type.—*PATHOLOGY.*—The hypertensive type may be primary, secondary or postoperative and affects children and young persons, seldom persons over 30. In general, the hypertensive type is caused by all the ear infections which produce the inflammatory type. However, there are some differences. The hypertensive type due to acute otitis develops in the third or fourth week, and often in the sixth or eighth week, of otitis, whereas the inflammatory type usually appears in the early phase of acute otitis. Observations of patients with hypertensive serous meningitis under medical care from the onset suggest that the acute otitis is always associated with fever of 100-104 F., indicating involvement of the dura or sinus. The slight infection of the meninges is a forerunner of hypertensive serous meningitis. The hypertensive type is not common with chronic otitis.

At times, the hypertensive type becomes manifest four to six weeks or longer after a simple mastoid operation. I believe that in these cases the operation was not adequate. In a considerable number of these cases pathologic changes were discovered in the sinus but exploration and drainage of the sinus were insufficient. The sinus infection was not halted, but progressed to serous meningitis which ultimately caused a hypertensive state. In others bilateral otitis was present and the less involved side was operated on, while the more involved side was either left

alone or was operated on when serous meningitis was already in progress. Occasionally a hypertensive state has developed when a simple mastoid operation was performed for chronic otitis.

The secondary hypertensive type is usually caused by an infection of the lateral sinus. It seems to be more common than the primary type, for in several cases of apparently primary type the sinus infection was unrecognized even during the mastoid operation. Several cases on record showed the entire, or almost entire, clinical syndrome of sinus thrombosis, but only a simple mastoid operation or no operation was performed and no chemotherapy was administered. Nevertheless, both the mastoiditis and the sinus infection showed spontaneous cure, to be followed in a few weeks by a hypertensive type of serous meningitis. More frequently the hypertensive type develops a few weeks after a sinus-jugular operation. In most of these cases the jugular vein on the right side, which usually carries more blood than the vein on the left (Fig. 6, p. 17), has been severed. This indicates that hypertensive serous meningitis not only is caused by inflammatory changes of the meninges but is probably encouraged by passive hyperemia. However, even in these instances the inflammatory exudate has a more important part than the transudate due to passive hyperemia, as proved by cases in which a sinus-jugular operation was done several years previously and hypertensive serous meningitis develops whenever the tympanic cavity becomes newly infected.

SYMPTOMATOLOGY.—According to our concept, the hypertensive type is one of the possible terminations of the inflammatory type. For this reason, patients presenting symptoms of the hypertensive type may also present several symptoms of the inflammatory type if active inflammation has not yet subsided. This explains the variation in symptomatology of the hypertensive state.

In hypertensive serous meningitis the mental state is clear. If the inflammatory and hypertensive phases overlap there may be some drowsiness, but conspicuous mental disturbances are not present. Vomiting and nausea are variable. Frontal headache of the bursting type is an outstanding symptom. Temperature,

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pulse rate and blood are normal unless the inflammatory phase has not yet disappeared. They are normal even in the hypertensive phase due to sinus thrombosis, since hypertension usually becomes manifest when the sinus thrombosis is cured either with or without surgery.

The principal symptom of hypertensive serous meningitis is papilledema, which may rise to 6 and 7 D. The rise is rapid and almost invariably associated with retinal hemorrhage. The degree of papilledema fluctuates and is usually different on the two sides. The visual fields do not show distinct alterations beyond a tendency to generalized restriction. Spinal puncture usually reveals lumbar pressure over 300 mm., but the cerebrospinal fluid is normal in every respect. The ventriculogram is normal; in some cases the ventricles are smaller than normal. The rachidial quotient of Ayala³ is more than 5. Hypersensitivity and motor and spinal cord symptoms are usually absent or not marked. Of the cranial nerves, the abducens is frequently involved on the side of the ear infection, on the opposite side or on both sides.

There may be difficulty in differentiating hypertensive serous meningitis and brain abscess in the quiescent phase. However, the patient's age, perfect mental alertness, high degree of papilledema and its rapid rise, normal blood count and normal encephalogram permit the diagnosis of hypertensive serous meningitis. With cerebral tumor the cerebral symptoms develop more gradually than with hypertensive serous meningitis unless there is hemorrhage into the tumor.

Prognosis.—Because hypertensive serous meningitis does not advance to purulent meningitis, there is no immediate danger to life. However, there is real danger to vision. Without treatment or if treatment is instituted too late, the papilledema may gradually subside, leaving various degrees of optic atrophy and caus-

³The rachidial quotient of Ayala (qr^a) is the ratio of the product of the final lumbar pressure (F) and the amount of cerebrospinal fluid removed (Qa) to the initial pressure (I), i.e., $\frac{Qa \times F}{I}$. With increased pressure of the cerebrospinal fluid, an index below 5 indicates a focal lesion, e.g., a brain abscess, an index above 5 is presumptive evidence of hypertensive serous meningitis. I have had no experience with this test.

ing impairment of vision and even amaurosis. If proper treatment is instituted early the prognosis is fair. However, the papilledema may not disappear for several months, and relapses, usually associated with a new infection, are not infrequent.

TREATMENT.—The first step is surgical removal of the focus of infection. Thereafter the purpose of treatment is to achieve decompression. Various methods serve that purpose. Some cases on record indicate that simple administration of laxatives decreased the papilledema. In others administration of hypertonic solutions has been successful.

Spinal puncture is highly important. Theoretically, it would be hazardous in the presence of high grade papilledema, but no ill effects have been reported provided the pulse rate was not diminished. Fluid is withdrawn very slowly; withdrawal must be stopped immediately if the patient complains of headache or if there is the slightest difficulty in breathing. Lumbar puncture is repeated the following day or in two days, depending on the patient's condition. In one of my cases papilledema of 6 D. disappeared after a mastoid operation, with exposure of the dura of the middle cranial fossa, and a few lumbar punctures. As long as papilledema of, say, 3-4 D. is not increasing and vision is not impaired, lumbar puncture may be continued for months. If, however, papilledema is increasing and the vision becomes slightly impaired, subtemporal decompression should be done. Cerebellar decompression should be done if there is clinical evidence of obstructive hydrocephalus, which is rare. Surgical decompression is the final hope for success in treatment, although even after this procedure relapses of the papilledema may occur. Moreover, after subtemporal decompression the brain may bulge through the opening in the skull for several years.

ACUTE PURULENT MENINGITIS

Acute purulent meningitis is a purulent inflammation of the pia-arachnoid which also involves the cerebral cortex.

Pathology.—The infection occurs by contiguity or continuity or by injury (p. 245).

Symptomatology.—This section deals particularly with the symptoms of the initial phase of meningitis; those of the advanced stages are discussed on page 268.

Acute purulent meningitis may be caused by (1) acute otitis media, (2) acute mastoiditis, (3) acute infections of the perilabyrinthine cells and petrous apex, (4) acute infections of the zygomatic cells, (5) chronic otitis media and (6) infections of the inner ear.

Acute infections of the tympanic cavity caused by a common upper respiratory infection may be the source of meningitis in any period of life. In infants and children the initial phase of acute otitis is frequently associated with striking systemic and cerebral symptoms, including fever, restlessness, delirium, convulsions and opisthotonos. These symptoms, which in adults definitely indicate a purulent infection of the meninges, gradually disappear after free drainage of the tympanic cavity is established. When they are present despite free drainage, they are alarming regardless of the patient's age unless they are caused by acute otitis on the opposite side. In infants acute otitis media caused by an upper respiratory infection is usually bilateral and usually more advanced on one side than on the other. Moreover, infant otitis is always of longer standing than the changes in the drum membrane indicate, so that the otitis on one side may be at its peak while the otitis on the other side is in the initial phase. For this reason, in the presence of a freely draining middle ear fever may be due to incipient acute otitis on the other side. If, however, the opposite side is definitely normal, fever of 102.2 F. and more may indicate incipient purulent meningitis and requires spinal puncture. Another symptom of incipient purulent meningitis is a decline in the child's vitality. The child becomes apathetic, sleeps much, although the sleep is not restful, eats and drinks poorly and does not resist medical examination. Objectively this decline in vitality is often manifested by an abrupt fall of temperature after paracentesis of the drum membrane. Purulent meningitis of infants usually originates in the tympanic cavity because the temporal pneumatic cell system is not developed.

In adults, infections of the tympanic cavity without surgical mastoiditis rarely lead to purulent meningitis except during epidemics of influenza, when meningitis may develop in the early stage of acute otitis before the formation of surgical mastoiditis. Formerly the diagnosis of *meningitis cum otitide* was made in these cases, indicating that the infection did not travel from the tympanic cavity into the meninges but invaded the tympanic cavity and meninges simultaneously. This concept is probably correct in many cases of meningitis in the early phase of acute otitis. In similar instances a careful history reveals that the otitis was of longer standing than the patient originally stated. Occasionally the meningitis is proved to have originated not in the tympanic cavity but in an infection of the sphenoid sinus or in pneumonia.

Purulent meningitis often originates in mastoiditis. There is, of course, a sagging of the superior and posterior walls of the external canal, but edema and tenderness at the tip of the mastoid frequently are not marked, nor is there necessarily a large amount of pus or extensive bone destruction in the mastoid process.

Purulent meningitis is especially important in old persons. Both mastoiditis and meningitis are often caused by pneumococcus type III and run a peculiar course. The otitis media causes typical mastoiditis in about four weeks. Unless a simple mastoid operation is performed, the mastoid symptoms decrease and the otitis passes into the subacute stage (p. 113) having a vague *symptomatology*. Occasionally the patient complains of deafness not relieved by air inflations, tinnitus, headache, dizziness, myalgias in the neck or the feeling "of having an ear." Unless these symptoms are properly evaluated, fulminating meningitis may suddenly appear, usually six to eight weeks after onset of the otitis. The outlook is extremely grave. Microscopic examination reveals that the infection has invaded the meninges through a deep perilabyrinthine cell, through the mesial wall of the mastoid antrum, through the tegmen tympani or, occasionally, through the posterior semicircular canal. In these instances the sul-

fanamides may have a disastrous masking effect if the surgeon is not familiar with the pathology. For this reason, in acute otitis of old persons, particularly with pneumococcus type III infections, sulfonamides should be administered only in the early stage. If not effective then they are seldom effective later.

Several otologists claim that purulent meningitis frequently originates in an infection of the perilabyrinthine cells (p. 111), particularly in old persons. A commonly accepted concept is that normally the temporal bone becomes more or less sclerotic in old age, whereas actually the normal temporal bone of old persons is usually well pneumatized. Owing to the extensive pneumatization, acute otitis in old persons is often more extensive than in young persons. For this reason, a simple mastoid operation in the aged is more extensive than in the young. If this operation is not thoroughly performed and if, for example, an inflamed pneumatic cell close to the dura is not opened, fulminating meningitis may follow. Persistent discharge from both the external canal and the retroauricular incision, slight rises of temperature, headache and x-ray evidence of inflamed pneumatic cells in the temporal bone are indications for re-operation. Chemotherapy is not effective.

Of the various types of chronic otitis, those involving the bony walls of the tympanic cavity may cause meningitis, particularly when associated with cholesteatoma. Statistics prove that chronic otitis without cholesteatoma causes meningitis more commonly between the twentieth and the thirtieth year, whereas cholesteatoma may cause meningitis at any age. Chronic infections of the tympanic mucosa without involvement of the bony walls cause meningitis about as frequently as they cause sinus thrombosis (p. 190). In chronic bone infections of the tympanic cavity an acute exacerbation is an important causative factor, and slight meningeal symptoms such as fever, headache and vomiting associated with an acute exacerbation strongly indicate the initial phase of meningitis. When due to an acute exacerbation the meningitis is usually caused by one organism, although the chronic otitis is a mixed infection. Another sign of incipient meningitis is mastoiditis in chronic otitis.

The mastoid symptoms in acute and chronic otitis are caused by different pathologic processes. In acute otitis the mastoid process is usually well pneumatized. The infection travels along the numerous anastomosing blood vessels between the mucosa of the mastoid cells and the periosteum of the mastoid cortex and external auditory canal, causing periostitis. This causes sagging of the superior and posterior wall of the external canal and tenderness and edema at the tip of the mastoid process. In chronic otitis the mastoid is not well pneumatized and there are few, or none, anastomosing blood vessels. Consequently, in a chronic case presenting mastoid symptoms, the periostitis is caused not by an infection which has traveled along anastomosing blood vessels but by extensive, destructive osteitis which has advanced to, or near, the periosteum at one or several microscopic sites. In these circumstances one can reasonably infer that the bone destruction has also advanced to, or close to, the dura, which serves as periosteum at the base of the skull. Thus mastoid symptoms with chronic otitis and a sclerotic mastoid process usually indicate the initial phase of an intracranial complication and require mastoid operation. Chemotherapy cannot replace surgery which, in this initial phase, usually achieves a definite cure. Rarely, meningitis occurs several years after a radical mastoid operation. The dura or sinus may or may not have been exposed at operation, but in all cases the postoperative cavity in the temporal bone contains newly formed meninges and adhesions which encourage retention of purulent exudate from an acute infection.

Of infections of the inner ear, diffuse purulent labyrinthitis may cause meningitis. Diagnosis is based on total deafness and nonexcitability on the caloric, turning and fistula tests. The labyrinthitis may be manifest or latent. When manifest there are vertigo, vomiting and spontaneous nystagmus of second to third degree to the opposite side; when latent there are mild vertigo and spontaneous nystagmus of first degree either to both sides or to the opposite side alone. The manifest type is most often caused by acute or subacute otitis media, and the latent type by chronic otitis, usually associated with cholesteatoma. Before the advent

of chemotherapy about 60 per cent of the cases of manifest diffuse labyrinthitis caused meningitis if surgery was not done, while in the latent type cerebellar abscess occurred in about 20 per cent and meningitis in only 5 per cent. The symptoms of incipient meningitis in labyrinthitis are fever, unilateral headache, tenderness of the cervical spine and slight rigidity of the neck on bending the head forward. Among these, headache and fever are of particular importance, although the temperature may not go over 99 or 100 F. In this phase the cerebrospinal fluid is clear, but there may be an increase of cells and globulins.

Meningitis caused by labyrinthitis invariably involves the posterior cranial fossa first. For this reason, the outlook is favorable because the meningitis remains localized for a comparatively long time (p. 259). Also the meningitis responds well to chemotherapy. Formerly, diffuse labyrinthitis with symptoms of incipient meningitis was an absolute indication for a labyrinth operation. Now, in a certain number of cases chemotherapy is as effective as surgery.

Clinical course—Otitic meningitis may run either a fulminating or a protracted course. The fulminating type appears suddenly, with numerous symptoms, including unconsciousness, delirium and Kernig's sign, and is usually fatal. The base of the brain especially is involved. The protracted type appears gradually, with indefinite symptoms, including malaise, headache, tinnitus and dizziness, for several days or weeks, until suddenly the typical symptoms of meningitis become evident. In other cases, particularly those due to mucous otitis, the meningitis may run an intermittent course over a period of weeks, similar to cysternal meningitis. The patient presents meningeal and cerebral symptoms which usually subside after mastoid surgery but reappear after several days and may subside again, until the generalized meningitis becomes evident. In a personal case the disease continued for almost five months after the onset of the original otitic infection.

The conspicuous focal brain symptoms, such as hemiparesis, jacksonian attacks, deviation of the eyes, paresis of lateral move-

ments of the eyes to one side and cortical facial paralysis, often lead to incorrect diagnosis of brain abscess. Encephalography may prevent unnecessary punctures of the brain. The prevalence of focal brain symptoms is due to particular involvement of the convexity of the brain. Autopsy usually discloses multiple patches of meningitis scattered over the convexity. Since microscopic examinations of the brain are not available, the pathogenesis of these meningeal patches is not exactly understood. Some otologists believe that the meningeal inflammation is more pronounced at the site of these patches than elsewhere in the brain, while others infer that the infection travels along the pial veins and that the meningeal patches are caused by local thrombosis or local emboli of the veins.

RHINOGENOUS MENINGITIS

ACUTE SEROUS MENINGITIS

Pathology.—Serous meningitis may be caused by acute purulent infections or by acute exacerbations of chronic infections in the frontal, ethmoid or sphenoid sinuses. Infections of the maxillary sinus apparently do not cause serous meningitis. Whether or not serous meningitis may originate in the nasal cavity cannot be stated.

Serous meningitis of rhinogenous origin is not as common as that of otitic origin. The sphenoid and frontal sinuses do not attain considerable extension before the sixth year. Infections of the ethmoid in young children more frequently involve the orbit than the meninges. For these reasons, serous meningitis of rhinogenous origin is not common in children, but serous meningitis of otitic origin is comparatively frequent. The latter type is often secondary, being caused particularly by sinus thrombosis, and, since infections of the paranasal sinuses seldom cause sinus thrombosis, serous meningitis caused by sinus thrombosis of rhinogenous origin must also be rare. Instead of sinus thrombosis, external pachymeningitis due to osteomyelitis is the principal source of serous meningitis of rhinogenous origin. The self-limiting type of acute osteomyelitis and chronic osteomyelitis are ap-

parently of great importance in this respect. Apparently serous meningitis also occurs in the spreading type of acute osteomyelitis, but the bone disease is so predominant that little attention is paid to eventual meningitis. Moreover, in spreading osteomyelitis serous meningitis is usually the forerunner of a purulent type and therefore never reaches the hypertensive state.

In the self-limiting type of acute osteomyelitis and in chronic osteomyelitis localized pachymeningitis externa often causes serous meningitis, probably by toxins which pass through the dura into the subarachnoid space (p. 99). This occurs most frequently when the posterior wall of the frontal sinus is involved. In these instances there is necrosis or a bone sequestrum which may or may not be adherent to the dura. The dura is hyperemic and covered by fibrin or granulations in subacute cases or is white or bluish red and markedly bulged in chronic cases. If the dura is incised, normal or hemorrhagic cerebrospinal fluid gushes from the subarachnoid space. Infrequently, the bone of the posterior wall of the frontal sinus is grossly normal. In infections of the ethmoid or sphenoid the clinical symptoms of serous meningitis usually disappear after surgery on the sinuses, so that the pathology is not known.

Inflammatory type.—The disease occurs in young people between 10 and 30 and occasionally in persons over 30. The symptoms are not as striking as in the inflammatory type of purulent serous meningitis. Nevertheless, the patients complain of headache which is more intense than that in common sinus infection and cannot be relieved by shrinkage of the nasal mucosa. The headache may cause hypersensitivity. There may be some apathy and sleepiness. The temperature may rise to 102 F. and over, and there is leukocytosis without shift to the left. The cerebrospinal fluid is normal or contains a few lymphocytes and a small amount of protein, causing slight cloudiness. The fundi may show passive hyperemia or a slight degree of papilledema. Rigidity of the neck, Kernig's sign, Babinski's reflex, ankle clonus, exaggerated tendon reflexes and, eventually, motor aphasia appear.

Treatment consists of drainage of the involved sinus and

chemotherapy. Radical surgery on the paranasal sinuses is not always necessary and particularly in children should be postponed as long as possible. A trial should be given chemotherapy and conservative measures, such as shrinkage of the mucosa, luxation of the middle turbinate, eventually resection of the anterior part of the middle turbinate, and lavage of the frontal or sphenoid sinus. If these procedures fail radical surgery should be considered. If serous meningitis is caused by osteomyelitis of self-limiting or chronic type, the sequestrum or necrotic bone must be removed; chemotherapy is useless in such cases.

Hypertensive type.—This type either appears gradually after an inflammatory phase or sets in with an acute manifestation of severe cerebral symptoms. The latter is usually seen in cases of serous meningitis due to frontal sinus infection. In these, epileptic attacks may occur, characterized by unconsciousness, deviation of the eyes, trismus, temporary cessation of breathing, dilatation of pupils and loss of reaction, tonic and clonic convulsions and tongue biting. After the attack there may be somnolence, bradycardia, vomiting, rigidity of the neck and papilledema. The cerebrospinal fluid is clear, but sometimes under increased pressure. Optical hallucinations and paranoid ideas are sometimes noted.

When the hypertensive phase is caused by an infection of the ethmoid or sphenoid, epileptic seizures are usually absent. There are intense headache, either supraorbital or occipital and radiating down the spine, and vomiting. In advanced cases there is papilledema of 3 D. or more; the cerebrospinal fluid is clear, occasionally contains traces of globulins and usually is under increased pressure. The Ayala index (p. 296) is more than 5. Patients frequently complain of dizziness and diplopia. There may be a positive Kernig sign, rigidity of the neck, diminution of reflexes, temporary hemiparesis on the side opposite the sinus infection, temporary aphasia and, with sphenoid infections, loss of pupillary reaction. Radical surgery on the involved sinus is essential to prevent amaurosis. If the operation is adequately performed the papilledema subsides very slowly, with optic atrophy occasionally persisting.

ACUTE PURULENT MENINGITIS

It is the consensus that rhinogenous meningitis is less common than otitic meningitis for reasons already discussed (p. 303). The former is seen more often in males than in females.

Meningitis may originate in acute infections or acute exacerbations of chronic infections of the paranasal sinuses or in infections of the nasal cavity. Apparently the usual source is the frontal sinus, less frequently the sphenoid and ethmoid and rarely the maxillary sinus. The nasal cavity is more often the site of origin than is usually assumed. The sphenoid sinus has special clinical significance. Some authorities state that it is involved in about 15 per cent of clinical cases of sinusitis and in 33 per cent of autopsy cases, but is responsible for approximately 35 per cent of all rhinogenous intracranial complications. In no one period of life is rhinogenous meningitis particularly likely to develop, but it is rare in early childhood unless it is caused by an orbital infection or osteomyelitis of the facial bones.

Rhinologic examination does not reveal changes suggestive of beginning meningitis; often there is no definite nasal pathology. With high fever the nasal mucosa is dry and the turbinates may be covered with crusts. Occasionally meningitis follows radical frontal sinus operations without being caused by surgical injury. A long period, up to 18 years, may elapse between the operation and the onset of meningitis. In these instances the surgeon failed to drain an infected temporal recess, or remnants of the sinus mucosa, allowed to regrow postoperatively, form a large cyst which later becomes infected and causes meningitis. Rhinogenous meningitis is usually caused by one organism, the commonest being streptococci, pneumococci and staphylococci. Infections with actinomyces are rare. Exceptionally, two or three strains of bacteria are present.

Frontal sinus.—Meningitis originating in the frontal sinus is either primary or secondary. It is primary when the infection travels through the posterior sinus wall. A large sinus favors the development of meningitis and other intracranial complications,

just as a highly pneumatized mastoid process is more frequently the source of an intracranial complication than a poorly pneumatized mastoid. Meningitis is secondary when the infection causes primarily another complication which in turn causes meningitis. Some authorities claim that, as in otitic meningitis, the secondary type is more frequent than the primary.

The primary infection is caused either by contiguity or by continuity. Infection by contiguity consists of osteitis which causes necrosis or a sequestrum of the posterior wall and external pachymeningitis. With spreading osteomyelitis originating in the frontal sinus, the infection spreads on the outside of the dura without invading the deeper layers. Less frequently, osteomyelitis involves only a small part of the frontal squama but immediately invades the deeper layers of the dura, causing subdural empyema and, ultimately, meningitis. It is impossible to predict the course the osteomyelitis will take. In infection by continuity the bone of the posterior wall of the frontal sinus appears grossly normal, but microscopic examination reveals infected thrombophlebitis of the anastomosing blood vessels. The dura, if exposed by surgery, appears to be normal, while its deeper layers may be inflamed, causing pachymeningitis interna and leptomeningitis.

The secondary type originates either in thrombophlebitis of the superior longitudinal sinus or in phlegmon of the orbit. For this reason, the symptoms of thrombosis or of orbital phlegmon, and eventually cavernous thrombosis, may mask those of meningitis.

In the primary type, symptoms suggesting incipient meningitis are headache, fever, photophobia, vomiting and orbital swelling. The headache is more intense than that due to infection of the frontal sinus; it increases at night and is not relieved by salicylates or by adequate drainage of the sinus. Fever may be due to the acute sinus infection if drainage is inadequate or to the basic disease, the common cold, which caused the frontal sinus infection. However, fever persisting despite proper drainage and after cure of the upper respiratory infection is a symptom which must

be properly evaluated, particularly if it continues despite chemotherapy. Frequent examination of the blood is required and spinal puncture should not be delayed. Photophobia and vomiting are definite meningeal symptoms and are indications for immediate spinal puncture.

The orbital symptoms are highly significant. If a frontal sinus infection causes meningitis the infection must travel through the posterior sinus wall. This wall has the same structure as the sinus floor, which is the roof of the orbit. Statistics prove that in frontal sinus infections the sinus floor is more frequently involved than any other wall. Consequently, it can reasonably be assumed that an infection which travels through the posterior wall will also travel through the inferior wall, but not vice versa. Therefore in a case of frontal sinusitis with vague symptoms of meningeal involvement, the symptoms of an orbital infection support the diagnosis of incipient meningitis. Administration of large doses of sulfonamides may obscure these observations: the orbital symptoms may subside but the drug fails to influence the meningeal infection.

A guarded prognosis must be given in all cases, particularly in meningitis of secondary type. In the primary type the outlook is favorable if the meningitis is caused by localized necrosis of the posterior wall.

Treatment is surgical and conservative; both methods are of equal importance. In the secondary type of meningitis conservative treatment is more effective than surgery. In the primary type all depends on whether the patient can undergo surgery and postoperative chemotherapy or requires the reverse order of treatment. If meningitis is incipient and the patient's condition good, an operation should be performed immediately, since the meningitis may be fulminating. Surgery is done by the external approach on the frontal and ethmoid sinuses. In the frontal sinus the posterior and inferior walls must be removed. The anterior wall should be removed if there is osteomyelitis or if the sinus is so large that inspection of the entire sinus is not feasible. Since in these instances the ethmoid, at least the anterior ethmoid, is al-

most invariably involved, an ethmoidectomy must also be done. This should be complete; i.e., both the anterior and the posterior ethmoid should be opened, even though only the anterior ethmoid is involved. The following case illustrates the danger of partial ethmoidectomy in purulent ethmoiditis.

In a case of severe frontal sinus infection, only the anterior ethmoid was opened. The operation was followed by fulminating osteitis of the rest of the ethmoid, of the nasal septum and of the frontal sinus and ethmoid on the opposite side. The infection did not spread to the meninges, but the patient died of rhinogenous septicemia, with multiple abscesses in the abdomen.

In meningitis caused by osteomyelitis of the frontal squama, surgery is absolutely necessary if the osteomyelitis is of the self-limiting type and there are sequestra. If osteomyelitis is of the spreading type, the management outlined on page 110 should be followed.

Ethmoid sinus.—Here too both primary and secondary types of meningitis must be considered. The primary type is caused by osteitis of the roof of the ethmoid or by infection passing along the anastomosing blood vessels through the roof. The secondary type is caused by cavernous thrombosis.

In the secondary type, symptoms of orbital phlegmon and cavernous thrombosis predominate. In the primary type, symptoms pointing to incipient meningitis are headache, fever, photophobia, vomiting and orbital involvement. The symptomatology is the same as in frontal sinus infections, but x-ray and clinical examinations prove the frontal sinus to be more or less normal.

Prognosis is the same as for meningitis originating in the frontal sinus.

Treatment is surgical and conservative. If surgery is performed, the external approach is preferable, because it permits better exposure of ethmoid cells which encroach on the roof of the orbit and exploration of the orbit.

Sphenoid sinus.—The primary type is caused by osteomyelitis of the sinus roof or by infection passing along the anastomosing blood vessels through the roof. The secondary type is caused by

cavernous thrombosis. In rare instances a patent cranio-pharyngeal canal (p. 78) serves as a pathway. In the secondary type the symptoms of cavernous thrombosis predominate. In the primary type, symptoms pointing to incipient meningitis are headache, most frequently localized in the occiput and perhaps radiating to the temporal region or down the spine, slight fever, photophobia, vomiting, pain in the ear and, occasionally, glycosuria. Orbital symptoms are usually absent unless the ethmoid is involved. Additional findings are difference in the size of the pupils, sluggish pupillary reaction and eventually absence of pupillary reaction; occasionally optic neuritis is noted.

For three reasons the prognosis is less favorable than for meningitis originating in the other sinuses. (1) The diagnosis of a sphenoid infection may be difficult, because frequently no pus is seen in the olfactory fissure or epipharynx and x-rays may fail to disclose involvement of the sphenoid sinus. Thus, the diagnosis is often made late. (2) The symptoms of incipient meningitis may be inconspicuous until suddenly the meningitis becomes manifest. This happens particularly in old persons, in whom diabetic coma or cerebral apoplexy is often diagnosed and only at autopsy is meningitis originating in a sphenoid sinus recognized. (3) Surgery is not as effective as in the other sinuses. Surgical treatment consists of endonasal opening of the sphenoid sinus and stripping of the mucosa. Exposure of the dura is not feasible. Some surgeons curet the marrow of the sphenoid body when there is osteomyelitis. I have never performed this operation. There is a possibility that curettage encourages spread of the osteomyelitis. Since these operations are not radical, conservative treatment is of utmost importance.

Maxillary sinus.—Primary meningitis probably does not occur, meningitis from infections of the maxillary sinus almost invariably being secondary. The infection either travels through the sinus roof and causes orbital phlegmon and cavernous thrombosis or travels through the posterior sinus wall, enters the pterygoid fossa and spreads along the blood vessels and nerve sheaths in the pterygomaxillary fossa toward the meninges. The infection may

cause osteomyelitis at the base of the skull, thrombosis of the middle meningeal artery, cavernous thrombosis and meningitis. Spread along the second pathway is not common; it occurs occasionally when an infected maxillary sinus is punctured and the needle perforates the posterior wall of the sinus. In meningitis originating in the maxillary sinus, surgery is useless; the only hope is in chemotherapy and the natural resistance of the patient.

Nasal cavity.—The importance of the nasal cavity as a source of meningitis is not fully appreciated, although instances of this type are by no means rare. The infection travels along the sheaths of the olfactory nerve which run through the foramina of the cribriform plate and carry the infection from the olfactory membrane into the subarachnoid space (Fig. 58). This follows surgical injury (p. 254). It must be strongly emphasized that in a case of definite infection of one or more paranasal sinuses, meningitis need not necessarily originate in the involved sinus, but may originate in an infection of the nasal cavity which travels along the olfactory bundles toward the meninges. The following case is an instructive illustration of this eventuality.

A boy, aged 16, had had severe headaches for two years. They were worse at night, often disturbing sleep. On June 12, after swimming and taking a sunbath, he had severe headache and fever. Symptoms increased until June 15, when he consulted a rhinologist, who diagnosed acute purulent infection of the right frontal sinus and applied displacement treatment. Neurologic examination gave negative results. The following day the patient had a chill. On June 18, when I first saw the patient, he was drowsy and temperature was 104.4 F. There were definite meningitis and left facial paralysis. Thick pus was noted in the right middle meatus and pharynx. The left side of the nose was normal. The blood showed 4,500,000 erythrocytes and 14,100 leukocytes. Spinal puncture yielded cloudy fluid containing many leukocytes but no bacteria. Surgery was considered too hazardous, so neoprontosil, hypertonic glucose solution, intraspinal air inflations and blood transfusion were given; penicillin was not available. On June 19, there were bilateral abducens paralysis and left hemiparesis. The patient died that evening, after an acute illness of seven days.

Autopsy revealed a large subdural abscess over the right cerebral hemisphere and basilar meningitis, although the cerebrospinal fluid

had not contained bacteria the day before death. The anterior portion of the sagittal sinus contained an obliterating thrombus. The other dural sinuses were normal. The right frontal sinus was filled with thick pus and the mucosa was brittle and mottled grayish black. There was hyperemia of the mucosa of the left frontal sinus and the

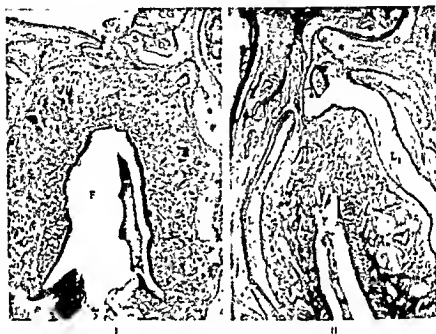


FIG. 85 - I, right nasal cavity. *F*, olfactory fissure, *M*, inflamed olfactory membrane, *B*, thrombosed blood vessels, *N*, olfactory fibers, *D*, roof of nasal cavity; *d*, congenital dehiscence through which dura (*G*) communicates with olfactory membrane, *b*, infected marrow space in roof of nasal cavity, *CG*, crista galli, *S*, sphenoid. II, same cavity. *F*, olfactory fissure, *M*, olfactory membrane, *N*, olfactory fibers, *L*, lymphatic vessels filled with serous exudate and leukocytes, *L'*, lymphatic vessels dilated by artefact, *N'*, olfactory fibers passing through cribriform plate and ensheathed by leukocytes, *d*, dura in olfactory rim, *S*, sphenoid, *M*, infected marrow space in crista galli (*CG*).

ethmoid and sphenoid sinuses. The sphenoid contained a little mucus. These findings pointed definitely to meningitis originating in infection of the right frontal sinus. Since there was no osteitis of the posterior wall of the frontal sinus it was assumed that the infection traveled along the anastomosing vessels from the sinus mucosa to the dura. Microscopic examination of the left frontal sinus revealed an acute inflammation of the mucosa and of the fibrous tis-

sue within some bone marrow spaces. In both ethmoids the mucosa was exceedingly edematous, but there was no destruction of bone. In the right frontal sinus the mucosa was inflamed. In the posterior wall there was mild osteoclasts along the margins of the blood vessels and the connective tissue of several of these channels was inflamed, but there was no pathway of infection from mucosa to dura along the blood vessels. The nasal cavity, however, showed severe inflammation of the olfactory bundles and the lymphatics of the olfactory membrane which run near the periosteum and parallel to the bundles; there was microscopic osteomyelitis of the cribriform plate, crista galli and orbital process of the frontal bone (Fig. 65). An interesting finding was a congenital dehiscence in the roof of the right olfactory fissure which failed to show signs of inflammation (Fig. 65).

This case proves clearly that meningitis may originate in the nasal cavity despite definite evidence of sinus infection. It also proves that in instances of this type surgery on the involved sinus is useless. A similar sequence of events may also occur in infections of the ethmoid, sphenoid or maxillary sinus. Acute infection of the nasal mucosa, in this case acquired by swimming, may be caused in other cases by a common cold or a minor endonasal operation.

The symptoms indicate a severe infection: headache, fever, chills, general malaise and, eventually, fulminating septicemia. The outlook is not favorable. Surgery in the area of the cribriform plate is not successful. Sulfonamides have not been effective in my experience, and whether penicillin will improve the outlook cannot yet be stated.

Considering the frequency of meningitis of nasal origin, it is important in a given case to determine whether the meningitis is due to an infection of the nasal cavity or of paranasal sinuses. Differentiation often can be based on orbital changes. Orbital swelling indicates that the infection originated in the frontal or ethmoid sinuses (Figs. 52 and 53), and surgery is eventually indicated. In an occasional case of meningitis which originated in the frontal sinus orbital symptoms may be absent, but even in these cases the patients complain of pain in the eye or give a history of orbital inflammation. On the other hand, in definite rhinog-

eous meningitis without orbital symptoms the infection probably traveled through the cribriform plate or roof of the sphenoid, or both. If clinical and x-ray examinations show a sphenoid infection, surgery on the sphenoid is indicated; if the sphenoid sinus is definitely normal, surgery is useless.

Clinical course.—Like otitic meningitis, rhinogenous meningitis may have either a fulminating or a protracted course. The former is commoner, particularly in cases of nasal origin, which, before the advent of chemotherapy, lasted eight or 10 days or less. I have not observed a protracted case. In reported cases the meningitis continued over a period of a year and occurred in chronic infections of the ethmoid or frontal sinus; the meningitis ran an intermittent course.

PHARYNGEAL MENINGITIS

ACUTE SEROUS MENINGITIS

A few cases on record supposedly prove that a hypertensive phase of serous meningitis may be caused by an infection of the tonsils or nasopharynx. Papilledema, increased cerebrospinal fluid pressure and a slight infection of the tonsils or nasopharynx preceded the symptoms of intracranial hypertension. In none of these cases was an inflammatory stage noted. I doubt that the hypertensive serous meningitis was caused by an infection of the pharynx and assume that there was merely a coincidental association of the hypertensive phase and pharyngeal infection. It is possible that a systemic infection such as influenza, a common cold or scarlet fever caused both the hypertensive state and the pharyngeal infection.

The pathogenesis is entirely different when a pharyngeal infection enters the pharyngomaxillary space and causes septicemia which, in turn, gives rise to serous meningitis. It is amazing that cases of this type are not more common, since serous meningitis is not rare in systemic infections. The serous meningitis in these cases is the inflammatory type. Whether there is also a hypertensive state is not known. The following case is an interesting example.

A woman, aged 43, had an infection of the left pharyngomaxillary space after tonsillitis. Three days after onset of the tonsillitis there was a picket-fence temperature curve, and two days later she became delirious and was hospitalized. There was swelling in the area of the left tonsil and soft palate, grimacing, dysarthria, abducens paralysis on the left and paresis and ataxia of the right arm and both legs. Tone and reflexes were diminished on both sides. Three days later the nodes in the left mandibular angle were swollen, but there was no trismus or tenderness along the sheaths of the neck vessels. There was marked swelling on the left tonsil, left palatopharyngeal fold and lateral wall of the pharynx on the left. No pathologic change was visible in the larynx. There was paresis of lateral movement of the eyes to the left and upward. Speech was conspicuously altered. The blood showed moderate leukocytosis and the cerebrospinal fluid showed a slightly increased globulin and albumin content. Operation was refused and chemotherapy was not available. The next day the swelling of the lateral wall of the pharynx increased, and there were convergent strabismus and a positive Kernig sign but no rigidity of the neck. The patient died in the evening.

Autopsy revealed a well encapsulated abscess the size of a walnut in the left pharyngomaxillary space. *Streptococcus haemolyticus* was found in the pus and blood. The spleen was enlarged, and there were signs of recent hemorrhagic nephritis and jaundice. The brain was hyperemic and edematous. Microscopic examination confirmed the diagnosis; there was no encephalitis. A diagnosis of encephalitis of the pons had been based on neurologic symptoms, when probably the symptoms were caused by brain edema due to pharyngeal septicemia.

ACUTE PURULENT MENINGITIS

A primary type of meningitis occurs when a phlegmon of the pharyngomaxillary space causes osteomyelitis of the sphenoid body which, in turn, involves the meninges by contiguity. Such cases are rare. When they do occur, they cause cavernous thrombosis first, then meningitis. The common type of pharyngeal meningitis is secondary to infections of the veins and large dural sinuses: the veins of the pterygoid and carotid plexus and the longitudinal superior, lateral and, particularly, the cavernous sinuses. The initial stage of meningitis is obscured by septicemia. The septicemia is not necessarily associated with high fever;

occasionally there are only slight rises of temperature, but the patient's general condition goes rapidly downhill. In any case, the patient is acutely ill, and symptoms of meningitis often become

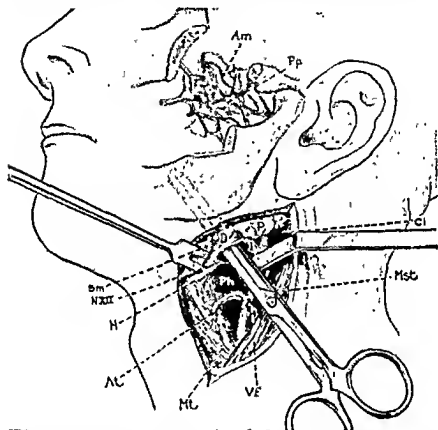


FIG. 60—Drainage of pharyngomaxillary space. *Am*, internal maxillary artery. *Ph*, pterygoid plexus. *ci*, internal carotid artery. *Mst*, sternocleidomastoid muscle. *VF*, vena facialis communis. *M*, sternohyoid muscle. *At*, superior thyroid artery. *H*, hyoid bone. *NXII*, hypoglossal nerve. *Sm*, submaxillary gland. *D*, digastric muscle. *P*, parotid gland. *c*, external carotid artery. *l*, lingual artery. *Ph*, pharyngeal wall. The dilator is introduced into the pharyngomaxillary space (By L. Bergmann).

evident after there is no hope of instituting effective treatment. Therefore the proper time for surgery is when symptoms indicate that the phlegmon of the pharyngomaxillary space has taken an ascending course (p. 220). Even in these instances chemotherapy without surgery might prevent the phlegmon from entering the

cranial cavity, but it is apparently safer to operate and then to give chemotherapy, because the intracranial complication may advance without causing alarming symptoms and chemotherapy may mask the insidious progress of the infection.

The operation, performed under ether anesthesia, consists of drainage of the pharyngomaxillary space (Fig. 66). The incision is made along the anterior border of the sternocleidomastoid muscle. The stylomandibular ligament between the parotid and submaxillary glands is perforated by a dilator introduced forcibly into the pharyngomaxillary space with the tip directed toward the tip of the nose. This is a simple operation when the anatomic landmarks are not obscured by pathologic changes. However, pathologic changes usually interfere. The lymph nodes are swollen, the connective tissue may be edematous, or there may be adhesions between the lymph nodes and salivary glands or sheath of the blood vessels. Occasionally the findings are so confusing that the submaxillary gland must be removed with the adherent lymph nodes in order to expose the stylomandibular ligament and pharyngomaxillary space. This, however, should not be a routine procedure.

If the anatomic landmarks are obscured the superficial fascia of the neck must be incised and the posterior belly of the digastric muscle exposed. The muscle is followed to its tendon, where it is perforated by the stylohyoid muscle. Above this muscle the pharyngomaxillary space can be entered. Retraction of the submaxillary gland forward in a submental direction removes the external maxillary artery from the surgical field. The sheath of the great vessels should be exposed extensively and adjacent lymph nodes removed. If the sheath is apparently normal, further exposure is not required. A necrotic sheath must be incised. Further procedure depends on the changes of the wall of the internal jugular vein. If it is inflamed or covered by granulations or if it is white and thickened but no thrombus can be felt, the tentative diagnosis should be periphlebitis. In this case there may be fever but no chills, spiking temperature or other symptoms of septicemia. Simple exposure of the area of periphlebitis and chemotherapy

will check advance of the infection. If clinical symptoms of septicaemia are present, the vein should be ligated below the involved area or the involved portion of the vein should be resected. Prophylactic mediastinotomy is seldom necessary because the purulent exudate rarely gravitates from the pharyngomaxillary space into the mediastinum.

Occasionally the infection of the pharyngomaxillary space involves the internal carotid artery. The prognosis is extremely gloomy because hemorrhages may occur into the pharynx or infected emboli may be carried into the brain, causing metastatic brain abscesses. The common carotid artery must be ligated.

TUBERCULOUS MENINGITIS SYMPTOMATOLOGY

Tuberculous meningitis may occur at any age but is most common in children during the first four years of life. Often there is an insidious onset, with preliminary symptoms including emotional instability, irritability, dislike of play, restless sleep, headache and nausea. This phase may last one to two weeks and is followed by a number of new symptoms, including intense headache, vomiting, constipation, dizziness, moderate rise of temperature and pulse rate and pain in the back, extremities and abdomen. These symptoms indicate the actual onset of infection. Several days later the infection becomes manifest, with positive Brudzinski, Kernig and Babinski signs, rigidity of the neck, convulsions, temporary stupor, difference in size of the pupils, photophobia, absence of knee jerks and abdominal reflexes, twitching and screaming. Fever may be remittent or intermittent. The pulse rate is frequently irregular in rhythm and force. About a week later the patient becomes comatose, the temperature is high and the pulse rapid and paralyses become more evident; there may be ptosis, strabismus and nystagmus, scaphoid abdomen and head retraction, and emaciation is marked. This phase may continue for several days, until death.

Deviations from this typical course are not uncommon. The course may be protracted or brief. When protracted the infection

may continue for two to five months. In such cases there may be only a few signs of meningeal irritation; the temperature may fluctuate between 99 and 100 F., and focal brain symptoms suggesting brain tumor may be the most important manifestations. In other cases of this type a remission for several days or several weeks is followed by reappearance of the meningeal symptoms and death. This protracted type occurs not infrequently in adults. When the course is fulminating there are no preliminary symptoms, but instead a sudden onset with somnolence and delirium, convulsions or aphasia and hemiplegia. The disease lasts only a few days, and in one case on record death occurred 30 hours after onset. Although the fulminating course occurs most often in young children, it also may occur in adults.

In a man, aged 54, with syphilis, tuberculous meningitis caused high fever, headache, backache, sleeplessness and stupor at the onset. Delirium developed rapidly, and in six days he died.

A woman, aged 40, had an acute exacerbation of chronic otitis on the left. She complained of dizziness and vomited frequently. Suddenly she became unconscious. On admission the left external auditory canal was filled with fetid pus and the drum membrane could not be visualized because of narrowing of the external canal. There were spontaneous second degree nystagmus, hyperalgesia and tenderness of both malar bones and the posterior cranial fossa. Tendon reflexes were absent; there was no Babinski reflex or ankle clonus. The left cheek was swollen and the skin cyanotic, and there was a draining sinus in front of the left tragus. Below the left ear were scars. The temperature was 103.4 F. A radical mastoid operation was performed, and a large area of necrosis was discovered in the region of the eustachian tube. Dura and leptomeninges were hyperemic. Three days after the operation the patient died, following an illness lasting about 10 days. Autopsy revealed miliary tuberculosis, tuberculoma of the left cerebral hemisphere and acute cerebral edema. Microscopic examination of the brain, which unquestionably would have revealed tuberculous meningitis, was not performed.

The cerebrospinal fluid in tuberculous meningitis presents the following features. Pressure is over 300 mm. of water at the onset but decreases in the phase of coma. The fluid is clear or like ground glass and colorless or faintly xanthochromic owing to

punctate meningeal hemorrhages or stasis. Very cloudy fluid indicates purulent meningitis. On standing a pellicle forms consisting of fibrin, cells and sometimes tubercle bacilli. The cells are increased in number, varying between 25 and 500 per cu. mm., with lymphocytes predominating. In the incipient stage the number of cells may be normal, although the protein content is increased from 45 to 500 mg. and the sugar and chloride contents are decreased. The protein increase is particularly marked if there is subarachnoid block, but there is no direct relation between protein content and progress of infection. The sugar level is below 45 mg per 100 cc. or, occasionally, is normal or, at the onset of the disease, even increased. The chloride content is strikingly reduced below 650 mg. per 100 cc. The lactic acid content is usually increased, and colloid gold test shows a mild midzone, or no, reaction. Tubercle bacilli are found in about 50 per cent of cases, although frequently most careful search of the pellicle is required. The blood may show moderate leukocytosis.

DIFFERENTIAL DIAGNOSIS

Regardless of the course the meningitis takes it sometimes interferes with the diagnosis of a common intracranial complication. I have considered tuberculous meningitis too often when the patient was a child and too rarely when the patient was an adult. Difficulty arises particularly in infections of the ear; tuberculous meningitis rarely interferes with the diagnosis of a rhinogenous complication. With ear infections associated with tuberculous meningitis, the following observations suggest correct diagnosis: the history of tuberculosis as given by the patient or his near relatives; the patient's age; the finding of tuberculosis in the body, particularly in the choroid, the large number of lymphocytes in the cerebrospinal fluid in the presence of progressive meningitis, and the eventual finding of Koch's bacilli in the cerebrospinal fluid. Frequently the ear findings are helpful. Purulent meningitis must be considered in the presence of an acute exacerbation of chronic otitis, acute otitis with surgical mastoiditis or a highly virulent acute infection characterized by profound deaf-

ss at the onset, persistent fever, involvement of the skin of the bony external canal and marked pain despite satisfactory drainage. If in the presence of increasing meningeal symptoms these ear findings are lacking, purulent meningitis is unlikely and more time should be taken for search for tuberculous foci.

Tuberculous meningitis occasionally causes symptoms of brain abscess, particularly in adults. Evaluation of the internal, neurologic and otologic findings will aid in correct diagnosis. If this fails, encephalography may be successful.

PROGNOSIS

Tuberculous meningitis is almost always fatal. Recovery is exceptional.

TREATMENT

In the presence of tuberculous meningitis surgery on the ear should be avoided because it may hasten death. Treatment is strictly conservative.

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Brain Abscess

PATHOLOGY

A BRAIN ABSCESS is a localized accumulation of purulent exudate in the white matter (subcortical abscess) or cortex (cortical abscess) of the brain.

Otorhinogenous abscesses are caused by (1) infection by contiguity and continuity, (2) metastasis and (3) injury.

According to Koerner's doctrine, that otitic brain abscesses develop at the site where the temporal bone infection comes in contact with the skull contents, otitic brain abscesses are located in the temporal lobe or cerebellum and rarely in the parietal or occipital lobe, whereas rhinogenous abscesses are located in the frontal lobe. A rhinogenous brain abscess is rarely located in the temporal lobe except with infections of extremely large sphenoid sinuses which extend far lateralward to the temporal lobe (Fig. 24, p. 74). Otorhinogenous abscesses are, therefore, in most instances "adjacent" brain abscesses.

Otorhinogenous abscesses are usually due to infection by both contiguity and continuity, with the infection traveling part of the way by contiguity and the rest of the way by continuity. Only brain abscesses originating in a sinus thrombosis are commonly due solely to infection by continuity. The thrombophlebitis spreads into and along the tributary veins of the sinus, with the infection invading the brain (p. 155). In this manner thrombophlebitis of the lateral sinus may cause an abscess in the cerebellum; less frequently it causes an abscess in the occipital lobe or

along the vein of Labbé (Fig. 7, p. 18) in the temporal lobe.

With a brain abscess originating in an infection of the tympanic cavity or paranasal sinuses the infection travels primarily through the bone, meninges and intermeningeal spaces to the brain. On passing through the bone and intermeningeal spaces the infection causes certain changes which for convenience may be called preliminary changes of brain abscess.

Preliminary changes.—These changes are important to the surgeon because they guide, and occasionally misguide, him in the search for a brain abscess. In a suspected case of brain abscess inspection of the bone and dura may disclose no gross pathology or slight or marked pathology. Evaluation of the findings is difficult. The finding of only slight changes at operation does not justify the inference that the pathologic process was slight during the entire course, for the changes may have been marked in the initial phase of the brain abscess and subsided spontaneously with further advance of the cerebral infection. On the other hand, striking changes such as subdural empyema or localized meningitis do not imply that they necessarily preceded formation of the brain abscess. They may be secondary, caused by expansion of the abscess toward the surface of the brain. For this reason, an analysis of the preliminary changes of otorhinogenic brain abscess is necessary, although hypotheses often must serve in place of actual knowledge.

An infection originating in the tympanic cavity or paranasal sinuses which is destined to enter the brain meets its first impediment in the bone. This may be overcome by infection by contiguity or by continuity. In the former instance the bone presents striking pathology—osteitis, necrosis or sequestration; in the latter the bone presents no gross changes. The incidence of one or the other finding, as noted by various surgeons, varies widely. For example, in my experience the bone was grossly normal in 70 per cent of cerebral abscesses and 75 per cent of cerebellar abscesses and was grossly involved in the rest of the cases. However, some statistics show exactly the opposite distribution. Apparently these statistics are based on a comparatively small num-

ber of cases and often lack the distinction between cerebral and cerebellar abscesses or between brain abscesses following acute or chronic otitis. The fact of practical importance is that the finding of grossly normal bone at operation does not exclude brain abscess.

To reach the dura the infection advances along one of two pathways: either it travels directly along the anastomosing blood vessels, i.e., by continuity, through the dura or it spreads between dura and bone. In the first instance the dura appears to be normal or hyperemic, but there is no external pachymeningitis. In the second instance there is definite external pachymeningitis and, eventually, an extradural abscess. Statistics concerning the frequency of external pachymeningitis vary considerably. In my experience external pachymeningitis was found with 56 per cent of cerebral abscesses and 64 per cent of cerebellar abscesses. Again it must be emphasized that other surgeons have reported exactly the opposite for reasons previously mentioned. However, the confusion is increased because many surgeons fail to differentiate external pachymeningitis in the area of the brain abscess from that remote from the abscess and active pachymeningitis from remnants of external pachymeningitis.

For example, in a case of brain abscess caused by rhinogenous osteomyelitis of the skull, there was extensive external pachymeningitis in the parietal lobe, but the brain abscess was discovered at the base of the frontal lobe beyond normal dura.

Also, there may be external pachymeningitis in the posterior cranial fossa and an abscess in the temporal lobe. External pachymeningitis remote from the brain abscess cannot be considered a preliminary change, and such cases were not included in my aforementioned statistics. Consequently, the surgeon should be aware (1) that the dura covering a brain abscess is often grossly normal and (2) that external pachymeningitis does not necessarily indicate a brain abscess in this area. The finding of normal dura, however, does not prove that there was never external pachymeningitis at this site. Brain abscesses caused exclusively or mainly by infection by contiguity may have a long period of

quiescence, during which external pachymeningitis may undergo spontaneous cure, leaving a thickening of the bone or dura, adhesions between bone and dura or, occasionally, grossly normal dura and bone. There is always external pachymeningitis if the bone is grossly involved, and there may also be external pachymeningitis even though at operation bone pathology is not noted in the area of the abscess.

The manner of spread of the infection from the outer layer of the dura to the pia is largely hypothetical. The infection may travel either by contiguity or along the blood vessels from the outer dural layer to the pia. The latter seems to be the principal route. In fact, infections which travel by contiguity from the external layer of the dura to the pia cause not actual brain abscesses but internal pachymeningitis and cortical ulcerations of the brain (cortical abscesses) if they reach the brain at all. In subcortical abscess the infection usually travels along the blood vessels through and beyond the dura, for this reason, dural fistula or necrosis does not occur. Consequently, if necrosis or a fistula of the dura is present, it is usually due either to subdural empyema or to expansion and rupture of a brain abscess (p. 341), but not to preliminary changes of brain abscess. Also, marked internal pachymeningitis with or without subdural empyema is not a preliminary change, but may be due to rupture of an abscess into the subdural space. Nevertheless, a mild infection of the internal layers of the dura and leptomeninges is quite in keeping with the general character of the preliminary changes. This mild infection may subside spontaneously or may leave adhesions between the dura and arachnoid or between dura, arachnoid and brain. When the infection has spread so far, the preliminary changes are completed and the way is open for invasion of the brain.

Formation of subcortical brain abscess.—Subcortical brain abscess is due to infection by continuity, the infection traveling from the leptomeninges along the blood vessels to the brain. Some authorities state that the spread may occur by perivascular infiltration, by venous thrombosis or by arteritis. This opinion is based on the microscopic examination of full-blown abscesses in

which conclusions concerning onset of the abscess are impossible. The few brain abscesses which have been examined in the incipient phase prove that the infection spreads along the veins and that the arterioles are rarely involved. The spread apparently occurs in the vascular sheaths as well as within the veins. Often the veins in the deep layer of the cortex and white matter present striking perivascular infiltration, whereas such infiltration is almost or wholly absent in veins elsewhere through the cere-

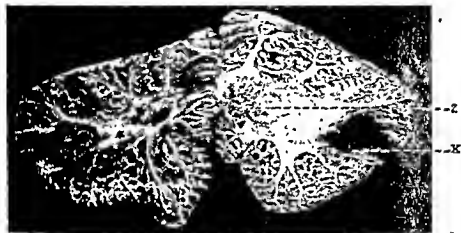


FIG. 67.—Cortical abscess of cerebellum (K). Z, dentate nucleus (After Grab-scheid.)

bral cortex. This indicates that the incipient phase of brain abscess primarily involves the white matter and eventually the deep cortical layers adjacent to the white matter, but not the rest of the cerebral cortex. The infection in the white matter causes localized encephalitis which is nonpurulent at first but soon becomes purulent if the infection is virulent.

In contrast with subcortical abscess, the cortical brain abscess (Fig. 67) is not actually a brain abscess but an ulceration of the cerebral or cerebellar cortex which may extend down to the white matter if the infection continues for a long time. Because the cerebral and cerebellar cortices have a rich blood supply (p. 81), a cortical abscess can be formed only with infections of long standing, such as protracted sinus thrombosis or internal pachymeningitis caused by chronic otitis. There is usually a fistula of the dura

or cerebral wall of the lateral sinus which leads into the cortical abscess. The abscess is surrounded by firm adhesions which are sequelae of an infection and which fasten together dura, leptomeninges and brain, but it is never encapsulated. A cortical abscess of this type may drain spontaneously into the lateral sinus

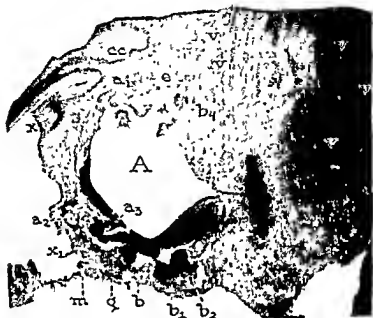


FIG. 68—Acute abscess of cerebellum (A) which at a_1 , a_2 and a_3 extends toward the cerebellar cortex (cc) and at b , b_1 , b_2 , b_3 , b_4 and b_5 extends toward the white matter. From x to x_1 there is no cerebellar cortex. The white matter is exposed and covered with pus. The secondary abscess (a_2) extends toward the granular zone of the cortex (g). m molecular layer of cerebellar cortex, e , purulent encephalitis, h , recent hemorrhage into white matter, v blood vessels with perivascular infiltration (nonpurulent encephalitis) remote from the abscess.

or mastoid and eventually undergoes spontaneous cure provided it does not extend too deeply into the white matter. Cortical abscesses are not common.

Expansion of acute brain abscess—A subcortical acute brain abscess expands primarily at the expense of the white matter. The inflammation spreads into the white matter and causes (1) small abscesses near the primary abscess cavity and (2) purulent or nonpurulent encephalitis which may spread over large areas

of the cerebellum or the involved cerebral lobe (Fig. 68). The small abscesses either displace or destroy the fibers of the white matter and ultimately merge with the original abscess, causing diverticula and pockets of the original abscess or become separated from it and form a second abscess cavity. The gray matter of the cortex, the corpus striatum and the dentate nucleus offer considerable resistance to the spreading infection, probably owing to a rich blood supply. However, gray matter covering the abscess which appears to be grossly normal may show microscopic evidence of degeneration and necrosis.

Contents of brain abscess.—The abscess contains pus which is creamy, or mucous and tenacious or crumbly. In some cases, particularly in anaerobic infections, the purulent exudate is extremely fetid and contains fragments of necrotic brain tissue. Usually streptococci or staphylococci are found, and occasionally pneumococci, *Bacillus coli*, *B. pyocyaneus*, *B. proteus*, *B. pyogenes foetidus*, meningococcus, gas-forming bacteria or *B. fusiformis*. In one cerebellar abscess I saw fusospirochetal organisms; the infection was rapidly progressing and did not show the slightest tendency to production of connective tissue. Search for anaerobic bacteria is of the utmost importance, particularly in brain abscesses caused by cholesteatoma of the tympanic cavity. Often the pus is reported to be sterile because of failure to search for anaerobic bacteria.

The purulent exudate of a chronic brain abscess caused by chronic otitis or sinusitis usually shows a mixed infection. Acute abscesses, particularly those caused by an acute infection of the ear or paranasal sinuses, commonly contain only one strain of micro-organism.

Walls of the brain abscess.—These consist either of necrotic brain tissue or of connective tissue. Necrotic brain tissue is found in acute abscesses and connective tissue in chronic abscesses. Microscopically the wall of an acute abscess presents various structures. Usually there is a layer of frank pus which gradually passes into an "infiltration zone." In this zone are protoplasmic glia cells, perivascular infiltration, eventually small abscesses, a

perivascular accumulation of fibrin, foci of necrosis and, occasionally, a few bacteria. The infiltration zone passes gradually into an area of nonpurulent encephalitis. These layers are evident at several sites of the abscess wall and not at other sites.

If the acute abscess is not fatal it may become chronic. In this case connective tissue forms in the infiltration zone and there is

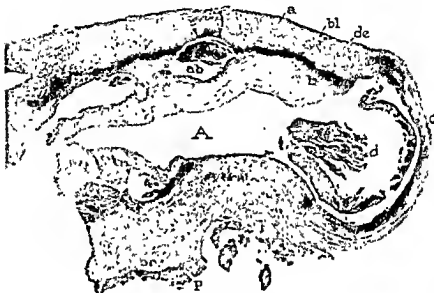


FIG. 69—Capsule of chronic temporal lobe abscess. A, abscess cavity, a, external layer of capsule, containing great number of blood vessels (bl), b, internal layer, which is necrotic, with portions released into cavity forming debris (d), de, demarcation between the layers, ab, small abscess in the capsule, br, necrotic brain tissue on outside of capsule, P, purulent exudate in an acute abscess surrounding the chronic abscess. (Compare with Fig. 71.) At c the capsule is thin.

minor proliferation of glia. These tissues ultimately form a capsule around the abscess. There is experimental evidence that encapsulation begins in the cerebral cortex or near it in the white matter and occurs last in the deepest segment of the abscess near the ventricle. The capsule (Fig. 69) may be 1 cm. thick at some sites, while consisting of only a few fibers near the ventricle. Presence of a capsule does not indicate definite cure of a brain abscess or even definite interruption of the inflammation. This is particularly true in otorhinogenous brain abscesses. In metastatic abscesses

e micro-organisms may lose their virulence, in which event the psule may become calcified. This almost never occurs in oto-inogenous abscesses, in which the capsule is not a stable structure. There are almost continuous changes both inside and outside the capsule (Fig. 69). The purulent exudate may invade the inner layer of the capsule, causing necrosis and rendering the

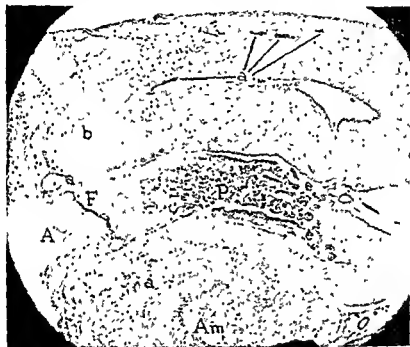


FIG. 70.—Ventricular fistula in temporal lobe abscess (A) F, fibrin barrier, P, choroid plexus; e, purulent exudate in inferior horn of ventricle, a, blood vessel with perivascular infiltration, b, brain edema, Am, cornu Ammonis.

capsule thinner and the abscess cavity larger. Thus, the chronic abscess expands by gradual outward shifting of the capsule, and owing to this type of expansion loculation is less common in chronic than in acute abscesses. Frequently the necrosis involves not only the inner layer but the entire thickness of the capsule. In this event a fistula forms, and the purulent exudate leaks either into the ventricle or into the subdural and/or subarachnoid space. In the first instance there is a "ventricular fistula" (Figs. 70, 84 and 88) and in the second a "brain-dura fistula" (Fig. 71). The

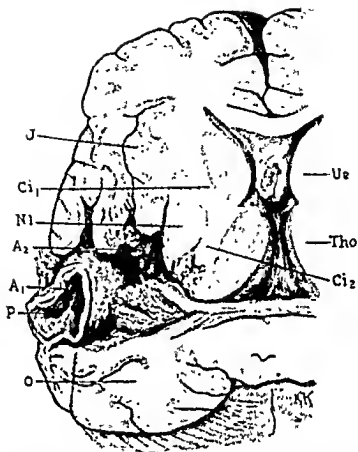


FIG. 71—Horizontal section through left cerebral hemisphere, temporal lobe contains chronic encapsulated abscess (A_1) surrounded by acute abscess (A_2). P , brain-dura fistula draining chronic abscess into tympanic cavity, O , occipital lobe, Nl , lentiform nucleus, Ci_1 , internal capsule (anterior portion), J , island of Reil, Tho , optic thalamus, Ci_2 , internal capsule (posterior portion), Ue , ependymal incisure.

inflammation may also extend along the blood vessels from the inner to the outer layer of the capsule (Fig. 69) if the micro-organisms in the abscess are highly virulent. Inflammation of the outer layer may serve as a source of infection which spreads into the surrounding brain tissue, causing brain edema, nonpurulent encephalitis or an acute abscess. In the last instance the acute abscess incorporates the chronic encapsulated abscess (Fig. 71).

To sum up, acute abscesses spread like a phlegmon, whereas chronic abscesses expand like certain types of brain tumors. For this reason, chronic cerebral abscesses frequently cause symptoms of intracranial hypertension, particularly decrease in size of the ipsilateral ventricle and increase in size of the contralateral ventricle. The contralateral hydrocephalus is probably caused by pressure against the midbrain, which cannot be displaced because of the incisura tentorii. For this reason, fluid escapes through the sylvian aqueduct only if ventricular pressure rises considerably. This backpressure acts equally in the ventricles on the two sides, causing ventricular dilatation. But on the involved side the increased pressure caused by the chronic brain abscess counteracts the tendency to dilatation of the ipsilateral ventricle, while the lower intracranial pressure allows dilatation of the contralateral ventricle. In the same manner, in chronic cerebellar abscess the ventricles on both sides become dilated (p. 55). Not infrequently the pressure on the midbrain causes a cutting of the cerebral peduncle of the opposite side by the sharp edge of the incisura tentorii. This may result in pyramidal symptoms, including paresis of the extremities on the ipsilateral side.

There is a question regarding the time required for formation of a grossly visible capsule. Some authorities claim that a capsule can be noted about 17 days after the infection of the brain, while others state that the formation of a capsule 2-8 mm. thick requires seven to ten weeks. In otorhinogenous abscesses a dogmatic statement regarding the stage of encapsulation to be expected at any given period of the infection is impossible. Some acute abscesses do not present the slightest microscopic evidence of capsule formation even though, according to clinical symptoms,

the abscess has been present for four or five weeks. Other abscesses of the same duration may present a definite capsule, at least near the cerebral cortex.

The formation of a capsule is the result of the action of bacteria on the brain tissue. Therefore capsule formation depends on (1) the general resistance of the host, (2) the type and virulence of the bacteria and (3) the underlying infection of the tympanic cavity or paranasal sinuses. Resistance is an intangible factor not easily measured and difficult to evaluate. As for the bacteria, some authorities claim that anaerobic and gram-negative bacteria are poor capsule-formers. However, this does not indicate that the action of aerobic and gram-positive bacteria favors capsule formation in all circumstances. Bacterial virulence varies, so that the same strain may cause different tissue reactions in the brain. The underlying bone infection also has importance in capsule formation. For example, if subacute otitis causing a brain abscess undergoes spontaneous cure, in due time the tympanic infection will no longer interfere with the formation of a capsule. On the other hand, if a cholesteatoma of the tympanic cavity has caused a brain abscess and in a short period one acute exacerbation is succeeded by another, each acute exacerbation indicates a new infection of the brain which interferes with formation of a capsule (Fig 77, p. 344).

Brain edema and fungus cerebri (herniation of brain).²—Because acute abscesses have no distinct limitations in the brain, they always cause inflammatory edema of the adjacent brain tissue (Fig. 72), although bacteria are found only in tissue near the abscess cavity. The edema may extend over large areas. Occasionally a comparatively small abscess causes edema of the entire ipsilateral hemisphere (p. 54). Brain edema is not as marked with chronic encapsulated abscesses as with acute abscesses and occurs only when there is active inflammation in the external layer of the

²Some surgeons distinguish between herniation and fungus of the brain. The former is considered to indicate a bulging of the brain which is covered by the dura, and the latter a bulging of brain tissue which is changed into a granulating and necrotic mass and is no longer covered by dura. I do not make this distinction because in brain abscesses brain tissue may bulge which is neither covered by dura nor changed into a granulating mass.



FIG. 72.—Left temporal lobe abscess T, temporal lobe, a, surgical opening of abscess. Note swelling of left temporal lobe, meningitis at base of left temporal and frontal lobes and ragged margins of opening of abscess owing to use of a dilator.

abscess capsule. Brain abscesses also cause spreading encephalitis, which may be nonpurulent or purulent. When purulent the encephalitis may lead to formation of a second abscess. Thus multiple abscesses form which are separated from one another by inflamed brain substance or by narrow channel containing necrotic brain substance. The encephalitis is more striking in acute than in chronic abscesses.

Both brain edema and encephalitis are of utmost importance in the formation of fungus cerebri (herniation of the brain). Fungus cerebri occurs when there are (1) an aperture in the bone and dura and (2) increased pressure of the brain. With otorhinogenous abscesses the aperture is caused almost solely by an operation. Punctures of the dura with a narrow cannula and nick incisions rarely permit the formation of fungus because the openings are too narrow. However, a large dural opening is not always required; occasionally an opening 1 cm. in diameter permits a herniation the size of a tangerine. On the other hand, as long as brain pressure is normal, even large incisions of the dura will not encourage herniation. Herniation occurs only with increased intracranial pressure, although papilledema may be absent and spinal pressure normal.

Increase of brain pressure may be caused by (1) ligation of the jugular vein, (2) hydrocephalus, (3) meningitis, both serous and purulent, (4) retention of pus in a brain abscess, (5) formation of a second brain abscess, (6) brain edema and (7) encephalitis. Ligation of the jugular vein in the absence of an obliterating thrombus in the lateral sinus increases intracranial pressure (p. 53) only temporarily and does not cause permanent herniation of the brain. Hydrocephalus causes permanent increase of brain pressure. For example, if a chronic cerebellar abscess causing bilateral hydrocephalus is incorrectly diagnosed as temporal lobe abscess, puncture of the temporal lobe will lead to herniation because of the hydrocephalus. Such cases are not common. Meningitis causes herniation, but the fungus does not grow as rapidly or reach such size as that due to brain abscess. A chronic abscess or retention of pus in a previously drained chronic

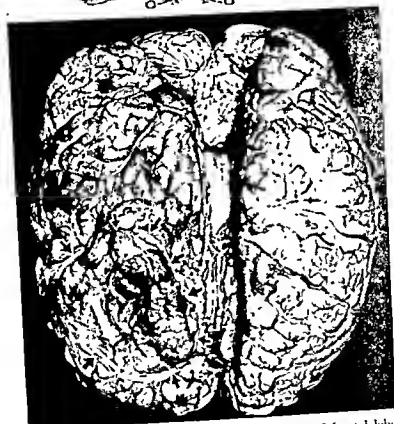


FIG. 73.—Below, herniation of brain (arrows) in case of frontal lobe abscess of rhinogenous origin, with abscess in most anterior part and in front of herniation. Brain viewed from above. Above, frontal section through anterior portion of herniation. *F*₁, superior frontal gyrus; *F*₂, middle frontal gyrus; *F*₃, inferior frontal gyrus; *c*, gyrus cinguli; *V*, anterior horn of lateral ventricle, *Cc*, corpus callosum; *Nc*, caudate nucleus; *r*, gyrus rectus; *o*, tractus olfactorius, *T*, temporal pole. Arrows indicate occipital wall of abscess. From *x* to *x*₁ the brain is softened due to the herniation. Owing to loss of fluids and preparation of the specimen, this part of the brain is shrunken.

abscess does not cause a large herniation unless there is encephalitis. The formation of a second brain abscess may or may not cause conspicuous herniation. However, multiple abscesses are not common in otorhinogenous infections, so that the most frequent causes of fungus with otorhinogenous abscesses are brain edema and/or encephalitis, the encephalitis being more important than the edema.

Herniation occurring immediately after incision of the dura is primary (Fig. 73), and that occurring one or several days after incision is secondary. Either may occur in otorhinogenous abscesses. The primary type is caused especially by acute abscesses. The pressure causing a primary herniation may be so powerful as to cause a bursting of the cortex of the protruding brain. Herniation involves the cerebrum more often than the cerebellum, probably because the edematous cerebellum can expand toward the basilar cisternae and foramen magnum, thus releasing the intracranial hypertension.

Fungus cerebri may steadily extend, become necrotic, become stationary or recede spontaneously. If it extends it may pull on the rest of the brain, particularly the ventricles, so that a ventricle bulges into the fungus. If the fungus becomes necrotic the ventricle may rupture toward the surface, with establishment of a ventricular fistula. This occurs particularly when part of the fungus has been removed by surgery. The pulling force of a brain herniation is well demonstrated in the following case.

In a case of rhinogenous osteomyelitis and frontal lobe abscess a large part of the frontal squama was removed. Later part of the parietal bone was removed, and at this site the dura was incised. An enormous primary herniation originated in the parietal lobe and developed so rapidly that the skin of the forehead on the opposite side was retracted (Fig. 74). After partial removal of the fungus the ventricle ruptured through the remainder of the herniation.

Among the causes of extension of brain herniation are passive hyperemia, spreading encephalitis and additional infection. When brain tissue protrudes through the dura and skull, the blood vessels of the brain must also protrude. If intracranial pres-

sure is high and/or the opening of the dura narrow, the veins which convey the blood from the fungus may be compressed. This results in venous hyperemia of, and hemorrhages into, the herniation. Also, the herniation may be increased by eucephalitis which spreads into the fungus and, eventually, into the diver-



FIG. 74—Herniation of brain in case of frontal lobe abscess and osteomyelitis of skull. Note epidermis growing over base of herniation and retraction of skin on right side of forehead, where the frontal sinus had been completely removed at a previous operation.

ticulum of the ventricle bulging into the fungus. If the eucephalitis persists for some time the brain tissue is replaced by edematous granulation tissue which may extend to the molecular layer of the cerebral cortex. Ultimately the granulation tissue penetrates this layer and appears at the brain surface (Fig. 75). Brain herniation also enlarges if an infection originating in the tympanic cavity or paranasal sinuses or at the surface of the herniation invades the fungus. If the infection remains at the surface of the herniation, the granulations covering the herniation become necrotic, with discharge of fetid pus. If the infection invades the depth of the herniation, abscesses may develop within the fungus.

Necrosis of the herniation occurs if a large part is destroyed by hemorrhages or if not only the veins but also the arteries which convey blood to the fungus are compressed. In this case large parts of the herniation fall off, but are rapidly replaced by other brain tissue as long as the pressure is increased. In rare instances

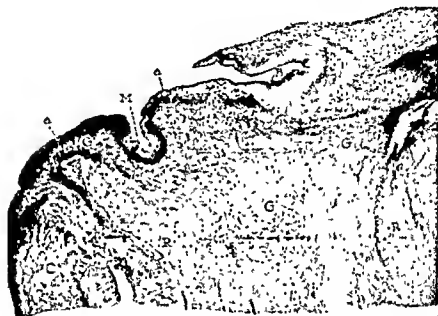


FIG. 75.—Section through brain herniation of over two months' duration. *C*, capsule of brain abscess, *G*, granulation tissue, *M*, molecular layer of cerebral cortex; *B*, bacteria covering cerebral cortex, *R*, cerebral cortex. Granulation tissue advances toward and replaces the molecular layer to reach the brain surface.

tissue of the herniation becomes mummified and falls off. With cure of the brain abscess and encephalitis the fungus cerebri recedes spontaneously (Fig. 76, see also Fig. 87, p. 395). The surface is covered by granulations which originate at the margins of the defect of the skull and dura or emerge from within the herniation. The granulations gradually turn into connective tissue, which extends into the dura and brain and is finally covered by epidermis. The bulk of the herniation also is changed into connective tissue, which replaces the destroyed white and gray matter. The loss of gray and white matter is seldom as considerable as the

size of the herniation would suggest, because the size is due largely to edema. If the opening in the dura is narrow and the fungus is large, the herniation may become permanent. In these instances the bulk of the herniation consists principally of con-



FIG. 76.—Subsiding brain herniation following temporal lobe abscess.

nective tissue containing a number of cysts filled with cerebrospinal fluid. A persistent herniation bulging into a mastoid cavity or a paranasal sinus may become infected, but such cases are not common.

Rupture of brain abscess.—As in abscesses in other parts of the body, the pus of a brain abscess tends to escape toward the surface or toward a pre-existing cavity. In cerebral abscesses, both acute and chronic, the purulent exudate may escape into the lateral ventricle or the meninges. In the first instance a ventricular

fistula (Fig. 70), and in the second a brain-dura fistula, is established. Abscesses of the temporal lobe usually burst into the inferior or posterior horn, while abscesses of the frontal lobe rupture into the anterior horn of the lateral ventricle. The acute abscess is likely to extend toward the ventricle, whereas a chronic cerebral abscess tends to expand toward the cerebral cortex. In fact, for a brain abscess to become chronic, a connective tissue barrier must prevent extension of the infection into the ventricle. Otherwise, the abscess bursts into the ventricle before capsule formation. The purulent exudate of cerebellar abscesses usually leaks into the meninges; rupture into the fourth ventricle is not common.

A ventricular fistula is caused by rupture of a brain abscess into the ventricle or by rupture of the ventricle into an abscess cavity. When an acute abscess approaches the ependyma of the ventricle there are hyperemia and exudation of fibrin in the choroid plexus. These changes cause formation of a plug, consisting of swollen villi of the choroid plexus and fibrin, which acts for a time as a barrier against the impending infection of the ventricle (Fig. 70). This action of the choroid plexus is supported by compression of the ventricle by the abscess. For this reason, rupture of the abscess seldom causes an immediate inundation of the cerebrospinal fluid with bacteria. In fact, rupture often fails to cause conspicuous changes in the cerebrospinal fluid. Apparently this is not an acute but a chronic process, whereas rupture of the ventricle into the abscess is distinctly acute. In some cases on record rupture of an abscess caused only localized empyema of the ventricles.

Obviously the protective mechanism of the choroid plexus must finally fail unless the abscess is surgically drained. With this failure, frank pus pours into the ventricle, causing pyocephalus. For this reason, rupture does not immediately cause alarming symptoms such as unconsciousness, general convulsions, high fever, rapid pulse, Cheyne-Stokes respiration, incontinence and vertical nystagmus. Nor is it immediately and invariably fatal. In my opinion all of these symptoms are caused instead by acute

brain edema which may or may not be associated with rupture of an abscess into the ventricle. Even the vertical nystagmus does not indicate rupture in all cases, since it may also be noted in cases of meningitis without brain abscess (p. 274). Favoring this concept is the fact that the aforementioned alarming symptoms occur particularly with cerebellar abscesses; yet a rupture of a cerebellar abscess into the fourth ventricle is rare.

Rupture of the ventricle into the abscess cavity occurs (1) if pressure in the ventricle is considerably higher than that in the abscess cavity and (2) if the ventricular walls have lost their normal resistance. It was mentioned previously that ventricular pressure may be considerably increased with cerebral abscess. However, a ventricle can hardly burst into an abscess cavity filled with pus. In fact, ventricular rupture usually occurs during or after surgical drainage of the abscess. Cerebrospinal fluid gushes into the abscess cavity. This should not be confused with the discharge of turbid fluid which may be noted when, after surgical drainage of the abscess, the discharge of pus has ceased. This fluid, which drains slowly from the abscess cavity, originates not in the ventricle but in the abscess wall by a process of generalized exudation. With rupture of the ventricle the x-ray film usually reveals a pneumocephalon (air in the ventricle, p. 363). The contralateral hydrocephalus decreases after rupture of the abscess. One must infer that there is always softening of the ventricular walls from brain edema. For this reason, ventricular ruptures usually occur with acute abscesses. It is unlikely that a thick capsule will yield to increased ventricular pressure. Rupture of the ventricle into the abscess cavity is not always fatal. In several cases of this type, reported both by other surgeons and by myself, cure followed even without chemotherapy. In these cases the ventricular fistula was probably narrowed by the brain edema and then was obliterated by the proliferation of glia and connective tissue.

The brain-dura fistula is more common with chronic than with acute abscesses. Although in acute abscesses the inflammation may also extend through the cerebral or cerebellar cortex to

cause a brain fistula, the fistula drains into the subdural space and does not extend through the dura. In chronic abscesses all the changes are more striking. An encapsulated abscess extending toward the cerebral cortex interferes with the blood circulation of the overlying cortex, which becomes anemic, while the veins of the pia are dilated. Simultaneously there is a chronic, localized in-



FIG. 77.—Section through tegmen tympani (*t*), dura (*D*) and capsule of a temporal lobe abscess (*C*). *p*, pneumatic cell below tegmen, *d*, dehiscence of tegmen, *a*, dural fistula. At *e* the abscess capsule is adherent to the thickened dura. (After Schmechel.)

flammation of the meninges, resulting in meningeal thickening and obliteration of the subdural and subarachnoid spaces. With advance of the brain abscess and encephalitis toward the cortex, either a brain-dura fistula or a transmeningeal fistula develops.

The pathogenesis of the brain-dura fistula is as follows. A localized area of the abscess capsule and of the overlying cerebral cortex becomes necrotic and breaks down, forming a fistula between the abscess and the subdural spaces. If the subdural spaces are not sealed off by adhesions, subdural empyema results which may perforate the dura (p. 139). If the subdural spaces are

sealed off, subdural empyema does not occur, but a localized area of the dura becomes necrotic and breaks down, establishing the brain-dura fistula. The pathogenesis of the transmeningeal fistula follows. Owing to encephalitis and to the expanding brain abscess the cerebral cortex overlying the abscess becomes atrophic and

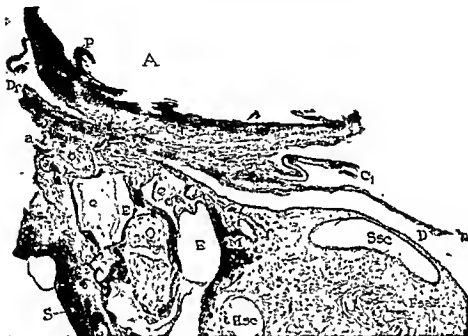


FIG. 78.—Section through chronic temporal lobe abscess (A), abscess capsule (C), dura (D) and tegmen tympani (t). S, Shrapnell's membrane, acutely inflamed, O, ossicles; E, epitympanum, M, mucosa of epitympanum, acutely inflamed; Hsc, horizontal semicircular canal, Ssc, superior semicircular canal, Fsa, subarcuate fossa; e, exudate in epitympanum; a, anastomosing blood vessel, D, thickened dura, P, purulent exudate; C, capsule, showing hyaline degeneration. At x, the capsule has united with the thickened dura above the tegmen, and there is no brain substance between capsule and dura. (After Schriener.)

vanishes. The abscess capsule then fuses with the thickened meninges (Figs. 77 and 78). Again a localized area of the capsule and meninges becomes necrotic and breaks down, establishing the transmeningeal fistula. Thus with a brain-dura fistula the cerebral cortex is pierced by the fistula, whereas with a transmeningeal fistula there is no cerebral cortex between abscess cavity and meninges. Since both fistulas are narrow channels running into the large abscess cavity, the condition is known as brain

abscess with stalk. According to this concept, these fistulas are the result, not the cause, of brain abscess. They exemplify an attempt of the abscess to drain into the adjacent bone cavity. They seem to form in the area where the infection entered the brain. Thus the fistulas are noted above the tegmen tympani in temporal lobe abscesses, behind the posterior wall of the frontal sinus in frontal lobe abscesses and in the area of Trantmann's triangle in cerebellar abscesses. Although the fistulas achieve some drainage, they do not accomplish a definite cure of the abscess, even though an additional fistula of the tegmen tympani, temporal squama or posterior wall of the frontal sinus allows pus to drain into the respective cavity.

Termination of brain abscess.—With successful drainage of the abscess the fibroblasts of the blood vessel walls and dura proliferate and produce connective tissue. In small abscesses the connective tissue fills the cavity and merges with the thickened meninges. There is also glia proliferation, but to a lesser degree. It is probably three to four months before a scar is definitely formed. The scar may be inconspicuous. In large and encapsulated abscesses the capsule collapses and the remaining cavity is filled with connective tissue which may harbor cystlike formations containing clear or yellowish fluid.

Metastatic brain abscess.—A metastatic brain abscess forms when, in the course of septicemia, infected emboli are conveyed from remote parts of the body to the brain. Only a few such abscesses concern the otolaryngologist. Rarely, a thrombus of the internal carotid in the temporal bone or pharyngomaxillary space gives rise to emboli which are carried into the brain. However, in these cases usually encephalomalacia, not a brain abscess, is the result. More important, although not common, are metastatic brain abscesses which originate in a thrombophlebitis of the large dural sinuses, particularly the lateral sinus. In these cases infected emboli are carried by the blood to sites in the brain remote from the sinus thrombosis (p. 155), causing purulent encephalitis of metastatic origin.

A woman with polyposis nasi and pansinusitis had two chills

during acute otitis on the left side and suddenly became unconscious. She died the day after an unsuccessful mastoid operation. Autopsy revealed empyema of the left frontal and maxillary sinuses and both sphenoid sinuses. There were purulent thrombosis of both cavernous sinuses and purulent thrombophlebitis of the left lateral sinus and left jugular vein. The brain showed purulent encephalitis consisting of miliary abscesses in the corpus callosum and in the lentiform nucleus on the right.

It is impossible to state whether the infection of the dural sinuses was caused by the otitis or by the infection of the paranasal sinuses, or both. Nor is it possible to state which dural sinus was the source of the septicemia. However, the appearance of the miliary abscesses and their localization in the gray matter indicated that the purulent encephalitis was metastatic in origin.

There is a commoner type of association of sinus thrombosis and brain abscess, although the brain abscess is not actually metastatic in origin. If in an obliterating sinus thrombosis the blood flow in the tributary cerebral and cerebellar veins comes to a standstill or is reversed, the inflammation may extend along the veins by continuity into the brain or fragments of the thrombus may be carried into the brain provided there is still circulating blood in the veins (p. 155). When sufficient infective material is conveyed into the white matter, a brain abscess may result. Thrombophlebitis of the lateral sinus or of the cavernous sinus is particularly likely to cause brain abscesses. Thrombophlebitis of the lateral sinus is not infrequently the source of abscesses in the cerebellar or temporal lobe but rarely causes abscesses in the occipital or parietal lobe. Subcortical cerebellar abscesses are usually caused by an infection of the inferior cerebellar veins, which are tributaries of the lateral sinus and run toward the inferior surface of the cerebellum. If an infection originating in the lateral sinus extends along the vein of Labbé, an abscess in the pole of the temporal lobe results. From the cavernous sinus the infection may extend along the middle cerebral veins to cause an abscess in either the temporal or the frontal lobe.

Traumatic brain abscess.—During puncture of the brain, if the cannula passes through the infected tympanic cavity or frontal

sinus and through the infected meninges an infection of the brain is possible. However, practical experience proves that this is extremely rare. The brain offers remarkable resistance to the common micro-organisms (in contrast with its susceptibility to toxins and the viruses) even though several punctures are performed. Experimental evidence also proves the resistance of the brain. Numerous experiments demonstrate that a brain abscess can be produced only if the brain is primarily traumatized and pure cultures of bacteria are injected into the necrotic tissue. However, this mechanism of infection is present in brain abscess following head injuries alone and is not related to surgical puncture.

Multiple brain abscesses.—Statistics indicate that multiple brain abscesses occur in 1.7–15 per cent of cases. Apparently these figures are not of great value because there are different types of multiple abscesses. Three types are comparatively common. (1) There are two or more acute or chronic abscesses in one part of the brain, e.g., in the temporal lobe. These abscesses are caused by expansion of one original abscess (p. 329). (2) There are two abscesses in one part of the brain, but one is chronic and encapsulated and the other acute and not encapsulated. The pathogenesis is discussed on page 333. (3) There are two abscesses in different parts of the brain, e.g., in the temporal lobe and in the cerebellum. Obviously the outlook is different for each type of multiple abscesses.

SYMPTOMATOLOGY

Clinical course.—Clinically, otorhinogenous brain abscesses are either acute or chronic. An acute abscess causes acute cerebral symptoms which continue or increase for several days and culminate in symptoms of generalized purulent meningitis. A chronic abscess also causes acute cerebral symptoms, but these subside after a few days, then return after a variable period and eventually cause purulent meningitis or paralysis of respiration. This definition presumes that no surgical treatment was applied.

It is customary to divide the course of chronic abscesses into

three or four phases: initial, quiescent, manifest and terminal phases. None is clinically well defined.

INITIAL PHASE.—This is easily determined in abscesses following head injuries or in metastatic abscesses, but it is often difficult or even impossible, particularly with chronic otitis, to determine the initial phase of otorhinogenous abscesses because there may or may not be striking symptoms. Usually the symptoms consist of fever, chills, headache, restlessness, dizziness, nausea, vomiting, focal brain symptoms and, eventually, slight meningeal symptoms. They may be inconspicuous and fugitive, so that the patient pays no attention to them, or they are striking and are misinterpreted as symptoms of incipient meningitis or sinus thrombosis.

The symptoms of the initial phase are essentially the same in both acute and chronic abscesses. The only difference is the time element. In acute abscesses the symptoms are usually striking and progressive or subside, say, for a day or two and then return with the same or increased intensity. In chronic abscesses the symptoms subside in a few days and may not reappear for as long as 15 months. The following case illustrates the acute type.

A woman, aged 41, had acute otitis on the right side which was apparently cured after two weeks. Two weeks later she suddenly became ill, with fever of 101.3 F. The following day she again complained of pain in the right ear radiating into the right side of the head and right mandible. There was continuous increase of headache, and eight days later there were dizziness and vomiting. Delirium gradually developed and eight days later the headache was almost unbearable. There were spontaneous nystagmus, facial paresis on the involved side and cerebellar symptoms. The temperature was only 99.3 F. Suddenly she became unconscious. The following day an operation was performed, but six hours later the patient died. Autopsy revealed a nonencapsulated cerebellar abscess on the right side and acute hydrocephalus, but no meningitis.

About four weeks after onset of acute otitis the first symptoms of brain abscess became evident. In 17 days the symptoms rapidly increased in quality and quantity to the time of death.

The following case illustrates the course of a chronic abscess.

A child, aged 4, had acute otitis which was apparently cured in about three weeks. Five weeks later the temperature rose suddenly to 101 F., with such dizziness that the child was not able to walk. Gradually fever and dizziness decreased, but she complained of intense headache and refused to leave her bed. Occasionally there was hydrocephalic cry. These symptoms continued for about three weeks. Then the temperature rose to 102.9 F. on three subsequent evenings, dropping to normal by morning. She complained of slight earache and intense headache on the right and dizziness. In the following three weeks the temperature again became normal, but symptoms indicated a tumor of the cerebellum. At the end of this time the child suddenly died. Autopsy revealed an encapsulated abscess of the right cerebellum and marked hydrocephalus, but no meningitis.

QUIESCENT PHASE.—During the initial phase it is impossible to predict whether the abscess will run an acute or chronic course. If it is destined to become chronic the initial phase is followed by the quiescent, or latent, phase. The term "quiescent" is preferable because there is seldom actual latency. The quiescent phase is ill defined. The symptoms may be striking or inconspicuous. They seem to be more marked when this phase is brief than when it is protracted. In all cases the temperature is normal, with perhaps an occasional sudden rise without tangible cause which continues for a day or two and then subsides. There are tachycardia, definite loss of weight and sleepiness. Nausea, vomiting and dizziness usually appear at the end of the quiescent phase. The eye grounds are normal or show slight changes. Papilledema was not common in my experience, except toward the end of the quiescent phase. The cerebrospinal fluid is normal or shows slight increase in protein and cell contents. The leukocyte count varies, often showing a slight increase.

The most significant symptom is headache, regardless of whether the quiescent phase is short or long. Evaluation of the headache is based on the type of infection in the tympanic cavity or frontal sinus (p. 100). Persistent headache in the fourth to sixth week or after spontaneous cure of acute otitis indicates an intracranial complication, although not necessarily a brain abscess. The diagnosis of brain tumor is often entertained until

postoperative examination reveals that what seemed to be a tumor is actually an encapsulated abscess. There is great risk for the patient if the erroneous diagnosis of allergy, migraine or head cold is made and the appropriate time for the operation is missed by mistaken administration of drugs. For this reason, an intracranial complication, particularly a quiescent brain abscess, should be considered whenever a patient complains of intense and persistent headache after acute otitis.

With infections of the frontal sinus certain features distinguish headache caused by the sinus infection from that caused by a brain abscess. The headache of frontal sinusitis makes the patient restless; that with frontal lobe abscess is associated with more or less marked drowsiness. With frontal sinusitis the headache is intense in the morning and decreases in the afternoon; with frontal lobe abscess it is almost continuous and increases at night. Salicylates, which relieve the headache of frontal sinusitis, do not influence that caused by a brain abscess. Headache caused by frontal sinusitis is associated with tenderness of the supra-orbital nerve, whereas that caused by a brain abscess may not be associated with tenderness of the nerve.

With chronic otitis, particularly with cholesteatoma, and with osteomyelitis of the skull the headache is usually caused by involvement of the dura (p. 101). With addition of a brain abscess the headache increases markedly, and general and focal brain symptoms indicate that not only the outer layer of the dura but also the brain is involved. Should these symptoms be absent, surgery will assist in making the correct diagnosis: headache caused by external pachymeningitis decreases after surgical exposure of the involved dura, whereas headache caused by a brain abscess does not subside unless the abscess is drained.

It can reasonably be assumed that during the quiescent period the abscess capsule is usually formed, regardless of whether there are few or many symptoms. In a brief quiescent phase rich in symptomatology, it is difficult to distinguish clinically between acute and chronic brain abscess, and a capsule may form even though the course is clinically similar to that of an acute abscess.

In many such cases the quiescent phase is followed immediately by the terminal phase unless there is surgical intervention. If, however, the quiescent stage is long and causes slight symptoms, a manifest phase appears before the terminal phase.

MANIFEST PHASE.—In this phase the temperature may rise abruptly or the patient suddenly becomes comatose. Paralysis of one leg, a jacksonian attack or a cerebellar convulsion may occur. The manifest phase appears either without apparent cause or after injury, including surgical trauma, excitement, gastrointestinal disturbances or acute infections. If the surgeon examines the patient for the first time in this phase and without knowing the exact history he may confuse the manifest with the initial phase, because they have a similar symptomatology. This error is disastrous if it leads to postponement of the operation, for the manifest stage is almost invariably followed by the terminal phase, in which surgery is futile.

TERMINAL PHASE.—This is characterized by symptoms of meningitis or respiratory paralysis.

The typical course of brain abscess is obscured if there is an additional intracranial complication such as sinus thrombosis. Even such evidence of general brain symptoms as papilledema and cerebrospinal fluid changes does not permit the diagnosis of brain abscess. Obviously the treatment of sinus thrombosis and septicemia is of primary importance, since in the presence of septicemia the cure of a brain abscess is not possible. After the septicemia is cured, the cerebral symptoms may gradually subside without further surgery. In these cases the cerebral symptoms are usually caused by serous meningitis, particularly in young persons. If, however, after the sinus operation systemic and cerebral symptoms persist or even increase, the diagnosis of brain abscess is justified.

Symptoms.—Brain abscesses cause different types of symptoms which may be conveniently divided into (1) systemic, (2) general brain, (3) aural and nasal and (4) focal brain symptoms. Only the systemic and general brain symptoms are discussed here, the others are discussed on pages 350, 401 and 411. The former

are the principal symptoms and are more important in the diagnosis of brain abscess than any others.

SYSTEMIC SYMPTOMS.—The patient is acutely ill and loses weight rapidly, even though appetite is good. The skin is flabby and the breath foul. There are intestinal disturbances, particularly constipation, and there is anorexia. Rarely, bulimia (excessive, morbid hunger) is noted. These symptoms cannot be caused by a simple infection of the tympanic cavity or paranasal sinuses and point strongly to cerebral involvement. The symptoms are striking in acute abscesses and are usually evident, although not marked, in the quiescent period of a chronic abscess. The rapid emaciation is of utmost importance.

Clinical experience indicates that the initial phase causes high fever and, eventually, chills for a few days. If an acute abscess forms, the temperature decreases slightly, staying between 100 and 103 F. until the terminal stage, when there is a striking rise, probably due to meningeal infection. If the abscess becomes encapsulated and enters the quiescent stage, the temperature decreases to normal or, particularly in cerebellar abscesses, even subnormal, except for occasional rises in the evening. The body temperature is an important guide to correct diagnosis of quiescent brain abscesses after a radical mastoid operation without drainage of the brain abscess. It is a common observation that immediately after a radical operation on the mastoid or paranasal sinuses the temperature may rise to 101 F. but usually disappears after an enema or the first dressing. If the temperature rises higher it is usually due to an infection of the surgical incision, incipient erysipelas or a quiescent brain abscess which was "awakened" by the operation on the bone. Infections of the skin incision and erysipelas respond to chemotherapy, but a brain abscess does not. With a brain abscess the high temperature may continue for one to three days after the radical operation. It may then be normal or almost normal for several days, until high fever reappears, ushering in the terminal phase. Chemotherapy is hazardous because it may lead to postponement of surgical drainage of the brain abscess.

The blood often shows a moderate increase of leukocytes. Eosinophils are usually present, occasionally in increased numbers. A shift to the left is common, but not marked. It has been emphasized that the leukocyte count becomes normal when the abscess passes into the quiescent phase. In a personal case in a child, aged 4, with a temporal lobe abscess, the leukocyte count was 5,400 (28 per cent polymorphonuclears, 5 per cent juvenile forms, 7 per cent eosinophils, 8 per cent monocytes, 52 per cent lymphocytes), although sulfonamides were not administered. However, a normal or subnormal leukocyte count does not justify the conclusion that the abscess is in the quiescent stage, because an acute abscess may cause the same leukocyte count. In a case of temporal lobe abscess which was fatal in the acute phase the blood count 20 days before death was 10,400 and 10 days before death only 7,100 (71 per cent polymorphonuclears, 1 per cent juvenile forms, 22 per cent lymphocytes, 5 per cent monocytes, 1 per cent eosinophils), even though no sulfonamides were administered. In a case of cerebellar abscess with sinus thrombosis and external pachymeningitis in a man, aged 33, the leukocyte count 13 days before death was 9,200 and six days before death only 7,800, although no sulfonamides were given. In phlegmonous encephalitis also, marked leukocytosis may be absent (p. 427). Chemotherapy makes the leukocyte count even less conclusive. The sedimentation rate is usually low. Peptonuria and glycosuria are seen occasionally.

GENERAL BRAIN SYMPTOMS.—Mental disturbances are of utmost importance. Since the classic description by MacEwen, "slow cerebration, heavy comprehension and marked want of sustained attention" have been considered significant of brain abscess. The patient is silent, the facial expression is depressed or apathic and there is compulsive yawning. Conversation is jumbled and speech drawing. The patient gives his name, address and age correctly after a short delay, he repeats the answer to the first question when a third and different question is put to him, he reads, but a few minutes later does not know what he was reading, and it is a great effort to perform small additions or subtractions in the

head. Sleepiness is striking, particularly in children, and in more advanced cases it is difficult to waken the patient. This condition may suddenly pass into coma. Some brain abscesses, both cerebral and cerebellar, cause symptoms of actual psychosis, resembling dementia, mania, alcoholic delirium and religious mania. In other cases no alterations of the mental state are present, and the patient's behavior is completely normal until he suddenly becomes comatose. The psychic symptoms do not permit conclusions concerning localization of the brain abscess.

Headache is usually continual. In the incipient phase headache is of moderate intensity; the patient complains of a band around the head or of heaviness in the head. Actions which cause venous hyperemia in the brain, such as coughing and straining at the stool, increase the headache. The intensity gradually increases and finally becomes almost unbearable. The patient groans and complains but is not as restless as a patient with meningitis. Rarely, headache is not present; this may occur in children with a quiescent brain abscess. Some patients localize headache in the area of the abscess, but others are unable to localize it. With frontal lobe abscess the headache is often localized in the forehead and temporal area; with cerebellar abscess there may be pain in the neck, eventually causing stiffness or opisthotonos. With temporal lobe abscess the headache may involve the temporal area, but often there is diffuse headache on the involved side of the skull.

Rigidity of the neck is not common with cerebral abscess, but is with cerebellar abscess. It may be associated with an abnormal position of head and frequent vomiting. In such a case spinal puncture should not be performed, because these symptoms usually indicate an impending cerebellar cone.

Nausea and vomiting usually occur, but are not repeated frequently. Nevertheless, in a patient who, for other reasons, is believed to have a brain abscess, vomiting unrelated to meals is an important symptom, even though it occurs only a few times. Nausea and vomiting are usually conspicuous with cerebellar abscess. Not only is there spontaneous vomiting, frequently with-

out preceding nausea, but there is reflex vomiting which can be elicited by the introduction of a tongue depressor or forceful opening of the mouth. This is particularly suggestive of cerebellar abscesses originating in sinus thrombosis, for in simple sinus thrombosis vomiting is not a conspicuous symptom.

Labyrinthine vertigo² is usually noted, but is not a principal symptom, even in cerebellar abscess, except those caused by purulent labyrinthitis. However, even in the last-mentioned cases vertigo is not marked at the time a cerebellar abscess becomes manifest. Often the patient complains of dizziness when he means headache or clouding of the visual field. Tenderness on percussion and pressure on the skull has no practical significance.

Slow pulse is not common with brain abscess. It is usually absent when intracranial hypertension is stationary, but appears with an acute rise of intracranial hypertension. Conclusions may be based on the pulse rate only when bradycardia is marked. There are cases on record in which operation was performed for brain abscess because the pulse rate was 50-55. At operation no abscess was found, and investigation revealed that the bradycardia was not caused by intracranial hypertension but was habitual. Bradycardia is occasionally noted with localized labyrinthitis with labyrinthine fistula and with diffuse serous labyrinthitis although there is no cerebellar abscess. With acute abscesses the pulse rate is usually related to the temperature except in the terminal stage, when there is often a sharp acceleration of pulse rate owing to vagus paralysis (Fig. 70). After successful drainage of a brain abscess tachycardia may be present for a long period.

Respiration is normal. However, as in brain tumor, paralysis of respiration may occur without warning, particularly in cerebellar abscess. Acute respiratory paralysis may be caused by simple movements of the body, such as rising from bed, stooping forward or straining at the stool, it may also be caused by spinal puncture or occur during operation on the mastoid or paranasal

²Labyrinthine vertigo consists either in the sensation of whirling (whirling vertigo) or in errors in recognition of the correct relationship between the person and space with reference to the sense of touch (tactile vertigo).

sinuses. In such cases only rapid drainage of the abscess will save the patient's life.

In a man, aged 42, a young physician performed a spinal puncture. The patient was not co-operative and was given a hypodermic injection of morphine. About an hour later 250 mg. of sodium pentothal was administered and, with the patient in lateral prone position, 7 cc. of clear cerebrospinal fluid was withdrawn. Spinal pressure was 400 mm. Ten minutes later respiration suddenly ceased, while the heart beat continued. The cerebellum was rapidly exposed and an abscess drained. However, respiration was not restored and despite artificial

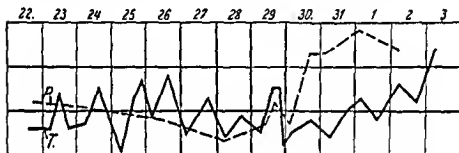


FIG. 79—Pulse rate (P) and temperature (T) in case of frontal lobe abscess. Note divergence of pulse rate and temperature five days before death.

respiration in the Drinker respirator for over 10 hours the heart finally failed. The eyegrounds were normal about 20 minutes before spinal puncture. Autopsy revealed a marked cerebellar cone.

The outcome was more fortunate in a boy, aged 9, with a cerebellar abscess. A simple mastoid operation was performed and the lateral sinus was exposed from the upper to the inferior knee. Suddenly jerking contractions of the sinus reduced the width of the sinus to nearly half its size; they were not synchronous with respiration. Shortly after the appearance of the contractions breathing ceased. The child was not cyanotic. There were clonic contractions of the floor of the mouth, but not of the neck muscles. The chest was fixed and the glottis wide open. The heart beat continued unchanged. Oxygen was administered, and after several minutes breathing was re-established. The operation was continued and the dura of the posterior cranial fossa exposed. Several minutes later the jerky contractions of the sinus reappeared and respiration ceased. The heart continued to beat and there was no cyanosis. Paralysis of respiration continued a little more than 25 minutes. Meanwhile the dura of the posterior fossa was further ex-

posed and the cerebellar abscess drained, although the circumstances did not permit operation under aseptic conditions. Immediately after drainage of the abscess respiration recommenced and recovery was uneventful. As in the preceding case, there was no papilledema.

The statistics on pathologic changes of the eyegrounds vary considerably. A summary suggests that in perhaps 50-60 per cent of cases of brain abscesses, both cerebral and cerebellar, there are pathologic changes of the eyegrounds. They usually consist of passive hyperemia of the retinal veins, tortuosity of the retinal vessels and blurred margins of the papilla. Usually the changes are more marked on the involved side and may become manifest comparatively late in the course. Papilledema is not common with either cerebral or cerebellar abscesses. I found papilledema in four of 22 cases of cerebral abscess (18.2 per cent) and in one of 12 cases of cerebellar abscess. The papilledema seldom exceeds 2-3 D., although statistics reported by neurosurgeons mention much higher figures. This is probably due to the fact that many cases seen by neurosurgeons were referred to them because of the papilledema. Despite papilledema vision is not often impaired. Exact examinations of the visual fields are not always feasible, and thus an enlargement of the blind spot or a slight concentric restriction of the fields may be missed. If the abscess is successfully drained, papilledema usually recedes in one to six weeks. If papilledema recurs it may be due to retention of pus in the abscess cavity, formation of a new abscess or, most frequently, serous meningitis. If vision was reduced before operation it is usually restored postoperatively. Permanent amaurosis caused by otorhinogenous brain abscesses is rare.

Papilledema is a favorable symptom because it usually occurs with chronic abscesses. This conforms with the concept that papilledema in brain abscess is caused by the intracranial hypertension, and not by toxins originating in the abscess. From this observation it has been concluded that the proper time to operate for a brain abscess is when papilledema is not progressing. No doubt this offers the best chance for cure because the abscess is well encapsulated and there is neither marked encephalitis nor

brain edema. However, for various reasons this concept is not acceptable to the otolaryngologist. (1) If operation were done only after papilledema had become stationary the mortality rate certainly would rise, because perhaps the majority of otorhinogenous brain abscesses do not continue for that length of time. (2) If a brain abscess causes papilledema there is always danger of respiratory paralysis, particularly if the papilledema is progressing. Therefore surgery is apparently more urgent in the presence of progressing papilledema than in cases of stationary papilledema. (3) I have seen at least three cerebral and three cerebellar abscesses which were well encapsulated and did not cause papilledema. Therefore, although the presence of papilledema certainly improves the outlook for surgical drainage of brain abscess, it is apparently hazardous to wait in all cases for papilledema, and particularly for stationary papilledema.

Spinal puncture is important in the diagnosis of brain abscess, but it is not without some hazards. As mentioned on page 61, it should not be performed in cerebellar abscesses. If symptoms indicate cerebellar abscess, the risk involved is greater than the additional knowledge gained by spinal puncture. In cerebral abscesses also, spinal puncture is not entirely harmless. I am not sure that a spinal puncture per se can cause rupture of the abscess (p. 61), but the psychic effects and forceful movements of the body induced by puncture may have a disastrous effect on the brain edema in the presence of intracranial hypertension. In the absence of symptoms of intracranial hypertension the risk is diminished, but certain precautions should be taken. Ether anesthesia and morphine should not be given. If sedation is necessary, bromides may be administered, and the puncture should be performed with the patient in the lateral prone position, unless cephalography is planned. If the spinal pressure is high, only 0.5-1 cc. of fluid should be withdrawn. If possible, frequent spinal punctures should be avoided except in cases of protruding brain herniation.

The findings obtained by spinal puncture are not uniform. Pressure is frequently increased regardless of the presence or

absence of papilledema. In other cases the pressure is normal. Decreased pressure is rare. The cerebrospinal fluid is usually cloudy, although in some cases of quiescent brain abscess it is clear. In a case of cerebellar abscess I found clear fluid one day before death. The cloudiness is caused by pleocytosis and increase of total protein content. The pleocytosis consists in an increase of the number of lymphocytes and polymorphonuclear leukocytes. Apparently the polymorphonuclear cells are more prevalent near the terminal phase, whereas in the quiescent phase lymphocytes predominate. Some surgeons have noted an increase of polymorphonuclear cells in the initial phase, an observation which I cannot confirm. The origin of the leukocytes in the cerebrospinal fluid with brain abscesses not associated with meningitis is not clear. Certainly pleocytosis does not necessarily indicate rupture of the abscess into the ventricle or intermeningeal spaces, because pleocytosis has been noted when autopsy failed to reveal any rupture of the abscess. Some authorities claim that pleocytosis is caused by the action of toxins on the meninges, while others believe that a toxic inflammation of the choroid plexus is responsible. I cannot make a definite statement, but experimental observations suggest that irritation of the choroid plexus is more important than irritation of the meninges.

The protein content is usually increased even when the fluid is clear, but the increase is not conspicuous as long as the meninges are not involved. The sugar and chloride contents are usually normal, occasionally even increased, provided there is no meningitis. If the fluid contains a large number of cells, the sugar content may be slightly decreased. The chloride content may be decreased if there is high fever or persistent vomiting. The absence of bacteria, at least in the quiescent stage, is important. Toward, or in, the terminal phase bacteria appear, indicating that meningitis is associated with the brain abscess. If in a case of brain abscess spinal punctures are performed frequently, one may occasionally note that the cell and protein contents decrease, although the patient's general condition is progressively worse. This antagonistic finding is called *syndrome de discordance* (p.

137). It is fairly indicative of *quiescent* brain abscess, although it may also occur in subdural empyema. The practical significance of this syndrome should not be doubted, but it should not be sought by performing frequent spinal punctures in all cases of potential brain abscess.

Recently x-ray examination has gained importance in the diagnosis of brain abscess. There are two methods, both associated with certain hazards. One consists of the filling of the ventricles with air, and the other of the filling of the abscess cavity with a contrast medium. The filling of the ventricles with air is accomplished by encephalography or ventriculography. The advantages and hazards of encephalography are discussed on page 65. For ventriculography, two small trephine holes about 1 cm. in diameter are made on both sides about 2.5 cm. from the sagittal suture and just above the coronary suture. A nick incision 2-3 mm. long is made in the dura and a cannula is introduced into the ventricles. Between 30 and 40 cc. of cerebrospinal fluid is withdrawn and an equal or slightly greater amount of air introduced. Since ventriculography may increase intracranial pressure, it should be performed only if everything is in readiness for immediate drainage of the abscess. In cerebellar abscess ventriculography is the only available x-ray method because lumbar encephalography is too hazardous.

The number of brain abscesses studied by air inflation of the ventricles is not large enough to permit definite conclusions concerning the typical findings. With temporal lobe abscesses the following observations have been recorded. (1) Complete or incomplete compression of the ventricle on the involved side. (2) Dilatation of the ventricle on the opposite side. (3) Lateral displacement of one or both ventricles toward the opposite side. This displacement involves particularly the anterior horn and body of the ventricle. The posterior portion of the cerebral hemisphere is, to some extent, protected against such dislocation by resistance of the falx cerebri and the union of the falx with the tentorium cerebelli. (4) Diminished filling of the subarachnoid spaces at the convexity on the involved side (Figs. 80 and 81).



FIG. 80 (*above*)—Encephalogram in case of temporal lobe abscess on left side, posteroanterior view. Note contralateral hydrocephalus.

FIG. 81 (*below*)—Contralateral hydrocephalus in case of temporal lobe abscess, lateral view. Inferior horns are not filled: *a*, ventricle on involved side, *b*, dilated ventricle on contralateral side.

With frontal lobe abscesses two observations have been recorded: (1) compression of the anterior horn downward on the involved side, and (2) dislocation of the ventricle on the involved side back and toward the opposite side. With abscesses of the occipital lobe a dislocation of the ventricle to the opposite side has been observed. The compression and shifting of the ventricles with cerebral abscesses are caused primarily by pressure of the brain abscess on the ventricles, and not by the associated encephalitis. This conclusion is based on observations in a case of phlegmonous encephalitis (p. 427). In that case there was only a slight compression but no shifting of the ventricles although the encephalitis caused a striking increase of brain pressure on the involved side. For this reason, I venture the opinion that pathologic changes in encephalograms are to be expected particularly with chronic brain abscesses, whereas with acute abscesses the findings probably are less conclusive.

I have not used ventriculography in brain abscesses. But as far as encephalography is concerned, its practical value, particularly in the quiescent stage, cannot be questioned. Not only is it useful in localizing the abscess; it is even more important in disclosing intracranial complications which cause the symptoms of brain abscess although there is no abscess. In this respect encephalography is frequently of decisive importance in serous meningitis, purulent meningitis of intermittent type (p. 302), progressive acute osteomyelitis of the skull and encephalitis. In all these diseases the encephalogram is normal and, therefore, may prevent unnecessary explorations of the brain. But despite this great advantage, in cases of brain abscess encephalography and ventriculography should be used only when all clinical and laboratory means are exhausted.

In a few recorded cases of abscesses of the temporal or frontal lobe, spontaneous pneumocephalon, i.e., a collection of air in the ventricles without previous air inflation, has been noted. In frontal lobe abscess air probably enters the abscess cavity through a brain-dura fistula and the ventricles through a ventricular fistula. Blowing the nose, sneezing and even normal respiration

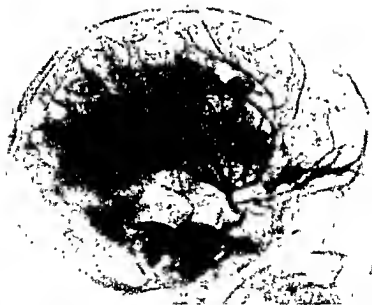
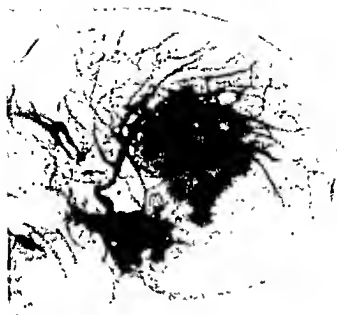


Fig. 82 - Above, right arteriogram of right temporal lobe abscess. The main middle cerebral arteries are displaced upward by the abscess (M). Below, same case, left arteriogram. The vessels run a straight course and are more widely separated from each other indicating hydrocephalus of the left lateral ventricle. (After Cairns and Jope.)

encourage the formation of pneumocephalon in these cases. The mechanism which allows the air to enter the ventricle in temporal lobe abscesses is not clear. The hypothesis has been advanced that the respiratory and pulsatile movements of the brain aspirate air into the abscess cavity and, if there is a ventricular fistula, into the ventricle. In this event, however, one would expect pneumocephalon to be more common than it actually is. In all cases of spontaneous pneumocephalon in temporal lobe abscesses thus far reported, air was discovered after injection of lipiodol into the abscess cavity, and it is possible that air was injected with the lipiodol into the abscess cavity or, exceptionally, into the subdural space.

To visualize the abscess cavity 2-5 cc. of 20 per cent iodipin has been injected into the cavity. Others use thorotrast, colloidal thorium dioxide or gauze soaked with 50 per cent abrodil. Others puncture the abscess and replace the purulent exudate by injection of iodipin and air. I have never used these procedures, because they irritate the meninges and increase brain pressure. Furthermore, if iodipin enters the ventricle, it may remain there for several months. Although no harm to the patient has been reported, these procedures should be used only when absolutely necessary.

The value of electroencephalography in brain abscess is discussed on page 82.

Arterial encephalography for the diagnosis of brain abscess consists of the injection of thorotrast into the common carotid artery. Immediately after the injection the skull is x-rayed. In several cases this method has furnished useful information (Fig. 82), but it can be routinely employed only if the proper equipment is available.

PROGNOSIS

Since spontaneous cure of otorhinogenous brain abscess is not proved, a practical discussion of prognosis can concern only cases treated by surgery. In these the prognosis depends chiefly on the following factors.

1. Formation of a capsule. There is no doubt that the presence of a capsule improves the prognosis considerably. Therefore, all factors which encourage the formation of a capsule (p. 334) improve the prognosis.

2. Association of another intracranial complication. When an abscess is associated with sinus thrombosis, marked meningeal inflammation or encephalitis, prognosis must be guarded. The prognosis is also impaired when there is an active infection in the underlying bony cavity. The infection in the bony cavity is a permanent, potential source of reinfection of the brain, particularly if dural fistulas communicate between the bony cavity and the brain (Fig. 77).

3. Time of diagnosis. Poor results are often due to delay or error in diagnosis.

4. Form of the abscess. Abscesses which form pockets and cavities require a guarded prognosis.

5. Location of the abscess. Cerebellar abscesses cause paralysis of the respiratory center more often than do abscesses of the cerebrum and, therefore, have a less favorable prognosis than do cerebral abscesses. Abscesses which extend close to the ventricle imply a greater danger than abscesses which are separated from the ventricle by a thick layer of normal brain tissue.

6. Time of operation. The outlook is best if the operation is performed in the quiescent phase, and particularly after acute otitis or sinusitis has undergone spontaneous cure.

7. Duration of postoperative treatment. It is an old clinical axiom that after surgery brain abscesses are cured rapidly or not at all. This is rather dogmatic but, to a certain extent, correct. After a brain abscess has been surgically drained, if discharge of purulent material continues over a period of weeks and is associated with unsatisfactory convalescence, one must conclude that there is progressive encephalitis and the patient's resistance is low. In these cases the outlook is not good.

The numerous factors which influence the prognosis for brain abscess render the outlook for operation doubtful. Even after a successful operation the prognosis is not necessarily favorable.

in every respect. The most important eventuality is intercurrent pneumonia or lung abscess. In fact, the mortality rate of brain abscesses must be attributed, to a considerable extent, to pulmonary complications rather than to infection of the brain.

Slight defects in the mental state and speech are not uncommon in children who recover from temporal lobe abscesses. More serious residua are epileptic seizures, particularly after recovery from frontal lobe abscesses. The attacks may be jacksonian or general and may appear several years after recovery. In several cases on record epileptic attacks were fatal. Autopsy showed the brain abscess to be completely cured, but there were foci of non-purulent inflammation near the scar. There are too few of such instances to permit satisfactory explanation of the epileptic attacks or of death. However, since after surgical drainage the collapsed capsule adherent to the meninges usually remains in the brain, it is amazing that epileptic attacks are not more common after cure of cerebral abscesses.

TREATMENT

The essential part of treatment is surgical. In the past, the operation was usually performed immediately after the diagnosis was established. The operation commonly consisted of a stab incision of the dura, puncture of the abscess with the knife, needle, cannula or special probe, forceful dilation of the incision and drainage by means of a rubber or glass tube. The results were not satisfactory, the mortality rate of cerebellar abscesses being close to 100 per cent in some clinics. Recently distinct progress was made when the operation was systematized. Unfortunately there is no uniformity of opinion. Therefore, the following discussion is not intended to offer a definite solution of the many problems involved; it presents rather the personal views of the writer which may or may not stand the test of time.

Time for operation.—It has been emphasized that operation should be done in the quiescent phase; in other words, operate only on chronic abscesses. This view is not entirely new. It is an old clinical observation that those abscesses have the best prog-

nosis which are discovered incidentally during an operation on the mastoid or paranasal sinuses, i.e., abscesses in the quiescent stage. However, one should not wait too long for the quiescent phase of otorhinogenous brain abscesses because many do not enter this phase. In these cases it has been recommended that conservative measures be used exclusively (p. 377). Such measures, however, will hardly achieve a cure of acute brain abscess. Whether penicillin will be more effective remains to be seen.

An acute otorhinogenous abscess must be considered inevitably fatal unless drainage is attempted. Granted that the mortality rate is high, operation apparently offers the only chance of saving the patient's life. Unfortunately, in the incipient phase there are no symptoms indicating whether or not the abscess will pass into the quiescent phase, but if symptoms suggestive of the initial phase do not subside or if they increase in two to four days, a quiescent phase is not likely to develop. In these circumstances exploration of the brain is apparently less hazardous than waiting for manifestation of the quiescent phase. In fact, in a case of acute brain abscess in which I operated and the patient died of pneumonia, autopsy revealed the abscess well drained by the operation. Nevertheless, operation should be delayed until the quiescent stage if the clinical condition permits postponement. In no circumstance should the operation be considered an emergency unless there is coma or difficulty in respiration.

Surgical field—It has been emphasized that operation through an infected field may cause an infection of the leptomeninges and/or the brain. There is obviously a difference between puncture of normal brain and puncture of a brain abscess through an infected field. If a brain abscess is drained and pus escapes, the surgical field can no longer be considered strictly sterile. It is probable that a certain degree of meningitis always follows abscess drainage. However, diffuse meningitis originating at the site of the puncture is extremely rare regardless of whether the operation is performed through a sterile or infected field. Osteomyelitis after operation through a sterile field is not as rare. In puncture of

normal brain, both clinical and experimental observations indicate that meningitis or brain abscess seldom follows.

The hazards of operation through an infected field are frequently exaggerated, whereas the advantages are not sufficiently emphasized. There are three particular advantages. (1) The abscess is drained where it is nearest the surface. All surgeons emphasize the importance of this factor. Since most otorhinogenous abscesses—except those caused by sinus thrombosis, are adjacent to the site of original infection, the approach through the originally infected cavity meets the abscess where it is closest to the surface. (2) Many otorhinogenous abscesses are associated with other infections, such as osteomyelitis, external pachymeningitis and sinus thrombosis. In an operation through the infected field such complications can be managed at the same time or, at least, are encountered in the same surgical field. This does not hold true for the operation through a sterile field. (3) In an operation through the infected field, a cerebral abscess is drained at the lowest point, a temporal lobe abscess through the tegmen tympani and a frontal lobe abscess through the floor of the frontal sinus. Thus, gravity influences drainage, whereas in an approach through a sterile field gravity does not come into play. In cerebellar abscesses drainage at the lowest point cannot yet be achieved.

There is no good reason for abandoning the operation through the infected field when there is active inflammation in the temporal bone or paranasal sinuses. In these instances a brain abscess is not a morbid entity but simply another intracranial complication, and it should be treated as such. The problem is different when a brain abscess follows acute otitis or sinusitis, i.e., after cure of the acute infection. In these instances a brain abscess is actually a morbid entity. Although even here operation through the mastoid or frontal sinus is suitable, there is no cogent reason for choosing this route. In addition, the scar through the mastoid or frontal sinus may be more conspicuous than one in the scalp. In comatose patients, when rapid action is required, operation through the sterile field may be preferable, although even in these patients it is unquestionably possible to establish drainage of the

brain abscess through the mastoid successfully and in due time.

Surgical technic.—Three methods are commonly used: (1) extirpation of the abscess, (2) the closed method, and (3) the open method.

EXTIRPATION.—The abscess is removed like a tumor. I have not used this method because it seems too hazardous for use in an infected field. Disregarding this hazard, the method is applicable only in a small number of otorhinogenous abscesses. In cerebellar abscesses it causes too rapid change of pressure in the posterior cranial fossa and should not be used. In rhinogenous frontal lobe abscesses, removal of part of the frontal pole of the brain is required to approach the abscess. In temporal lobe abscesses the method is applicable only if the capsule is quite thick and if the fibers of the capsule are not twisted with the nerve fibers of the brain. It may take three months or more for such a capsule to form, and this is too long to postpone operation for an otorhinogenous abscess, particularly when the underlying tympanic infection is chronic. It has been suggested that the abscess be punctured several times to prepare it for extirpation, but this is apparently a lengthy procedure fraught with hazards.

For operations through an infected field only the closed and open methods are available. The terms "closed" and "open" are only approximate. In the closed method the brain is punctured through a nick incision of the dura, whereas in the open method the brain is widely exposed before puncture. The practical significance of this terminology is that with the open method a brain herniation is quite likely to occur. With the closed method herniation is rare except in the presence of extremely high intracranial hypertension. The open method is believed to encourage infection of the subdural spaces, which does not occur with the closed method. In neither method is the *modus procedendi* uniform, regardless of whether the operation is performed through a sterile or an infected field. Since the operation through a sterile field is in the realm of the oentrosurgeon, this section deals only with the principles of surgery through an infected field.

The operation is performed under light ether anesthesia. The

increase of brain pressure caused by ether is so trifling that it does not interfere with the operation. In both the closed and the open method, wide exposure of the dura is the first step. Wide exposure is inevitable to remove all necrotic bone adjacent to the dura. But wide exposure should also be performed when the adjacent bone is normal, to permit search for localized pathologic changes of the dura and to allow arrest of hemorrhage caused by subsequent puncture of the brain. Up to this stage the procedure is the same for both the closed and the open method, except that in the open method the dura is more widely exposed.

CLOSED METHOD.—After exposure of the dura, a nick incision is made in the normal dura or, if there is external pachymeningitis, in the involved dura. Through the incision the brain is punctured (Fig. 93, p. 421). This is done with a blunt instrument, either a blunt brain needle or an open cannula with a rounded edge. There is experimental evidence that the open cannula injures the brain less than the blunt needle, although the difference is apparently not striking. However, it is an established fact that sharp instruments, particularly knives, should not be used, because hemorrhage in the subdural space and brain is frequently formidable, particularly if a spinal puncture is performed after brain puncture.

A capsule will resist the exploring cannula like a rubber ball. There may also be resistance although true encapsulation has not occurred, or no resistance despite encapsulation. The first type is difficult to explain, but there is microscopic evidence of such a finding. The cannula may not encounter resistance even with encapsulation when a chronic encapsulated abscess is surrounded by an acute abscess (Fig. 71, p. 332). In such a case the cannula may enter the acute abscess without resistance and does not come in contact with the capsule of the chronic abscess. Moreover, the capsule of an otorhinogenous abscess is often thin where the abscess is adjacent to the osteitic bone, i.e., at the stalk. Here the thin wall of the abscess is fused with the meninges, the brain substance between the membranes having been absorbed (Figs. 77 and 78), and the cannula does not pass through soft

tissue before reaching the capsule. If, however, in a case of this type the puncture is performed at a site remote from the stalk, a capsule may be encountered because the cannula passes through the soft brain tissue before reaching the capsule (Fig. 83). Since otorhinologists perform the puncture at the stalk and neurosur-

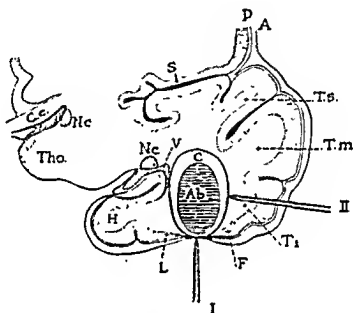


FIG. 81.—Puncture of temporal lobe abscess: *I*, through infected field, *II*, through sterile field. *Cc*, corpus callosum; *Nc*, caudate nucleus; *Tho*, optic thalamus; *H*, gyrus hippocampi; *L*, lingual gyrus; *V*, inferior horn of ventricle; *F*, fusiform gyrus; *Ti*, inferior temporal gyrus; *Tm*, middle temporal gyrus; *Ts*, superior temporal gyrus; *S*, sylvian fissure; *A*, arachnoid; *P*, cerebral cortex; *Ab*, abscess cavity; *C*, abscess capsule.

geons do it remote from the stalk, it is conceivable that neurosurgeons note a capsule more frequently than do otorhinologists.

After puncture the purulent exudate is aspirated. The further procedure varies: either puncture is repeated after a few days, or a tube is inserted in the abscess with the cannula as a guide. I prefer the first technique for two reasons. (1) Choice of the proper material for drainage is difficult. Tubes of rigid material are likely to injure the abscess walls and encourage rupture. Tubes of soft material are easily compressed by increased brain pressure and

are, therefore, ineffective. Strips of plain or iodoform gauze are apparently the best material for drainage, but it is difficult to introduce gauze through the nick incision. (2) In cerebellar abscesses drainage is scarcely feasible if the operation is performed through Trautmann's triangle. A soft rubber tube introduced through Trautmann's triangle almost invariably cracks and becomes useless. With a rigid tube, oblique drainage is established which does considerable harm to the brain tissue and is ineffective because a drainage tube into the brain should be perpendicular to the surface. Perpendicular introduction is easy in frontal and temporal lobe abscesses, but is hardly possible in cerebellar abscesses drained through Trautmann's triangle. For these reasons I use only puncture and aspiration in the closed method.

OPEN METHOD.—This can be performed in one or two stages. The dura is incised by a cross-incision or in stellate fashion, and the flaps are turned over or excised (Figs. 86 and 89). Several methods have been advocated to seal off the subdural spaces. Some surgeons encircle the dura by diathermy; others suture the meninges and cortex with interrupted catgut sutures and seal off the meninges and cortex by the electrocoagulating current, and others pack the subdural spaces with gauze saturated with a weak solution of iodine. Like many other surgeons, I have never had spreading leptomeningitis develop from the purulent exudate which escapes from a brain abscess and passes through the subdural spaces. There is probably considerable local resistance to the infecting organisms, and the leptomeningeal spaces immediately over the abscess are usually sealed off by adhesions or obliterated by brain edema. The local meningeal resistance may not be present if the operation is performed through a sterile field. When the operation is performed through an infected field, drainage is in the area which was exposed to infection in the initial phase of the abscess. This perhaps establishes a local immunity which combats any meningeal infection caused by the draining of the abscess. For these reasons, I believe that the sealing off of the subdural spaces is not necessary if surgery is done through an infected field, with chemotherapy pre- and postoperatively.

When the cerebral cortex is exposed some surgeons postpone drainage of the abscess for three to six days. In the meantime the abscess capsule, if smooth-walled and not bound down, will have migrated to the surface. This procedure is superfluous when the operation is performed through the infected field, because the abscess is always as near the surface as possible. Therefore, after exposure of the cortex it is immediately removed by electrosurgery or suction down to the capsule. This procedure is marsupialization or "unroofing of the abscess." Extracapsular necrotic and suppurative brain tissue which may be present can be removed by suction. After the overlying brain tissue is removed the capsule comes into view and the abscess is punctured. The outer presenting portion of the capsule is then removed in a circular manner to permit inspection of the entire interior of the abscess cavity. The cavity is filled with plain or iodoform gauze placed as a Mikulicz tampon. The gauze should be introduced gently and the cavity should not be firmly packed. In several cases on record the ventricle ruptured into the abscess cavity after firm packing of the cavity with gauze. To avoid the same accident, examination of the cavity with instruments or with the finger should be omitted. The floor of the cavity will now progress up toward the opening in the skull. Rapid herniation of the brain should be checked by repeated lumbar punctures, which can safely be done the third to fourth day after operation. If this is not possible and a large herniation forms, after-care extends over several weeks, but herniation does not necessarily render the outlook unfavorable.

The closed method causes little injury to the brain, and, for this reason, pareses and epileptic attacks rarely follow cure of a brain abscess. However, the closed method is not successful in abscesses with secondary diverticula, and there may be a recurrence after apparent cure. There is no recurrence after the open method. However, the open method traumatizes the brain tissue more than the closed method does and may cause considerable scar formation. For this reason, permanent pareses and, eventu-

ally, epileptic attacks may follow the open operation. They are not common if the operation is performed through the infected field, probably because surgery injures parts of the brain which are "mute" and remote from the central gyri.

After-care.—The after-care is as important as the operation itself. In the close method the bone cavity is loosely packed with gauze and the skin incision left open. If recovery is uneventful the first dressing is done two days after the operation and the gauze is replaced by new packing. The dressing is then changed daily until granulations fill the cavity. In the open method the bone cavity is also packed with gauze which is gradually removed in about a week if recovery is uneventful.

Convalescence may be disturbed by brain herniation, brain edema, encephalitis, refilling of the original abscess cavity, formation of a new abscess, meningitis or a pulmonary complication. With the closed method brain herniation is not common, but any of the other complications may occur. Systemic, general and focal brain symptoms, particularly mental disturbances, headache, papilledema and eventually convulsions, will appear or reappear, and convalescence will be retarded. Unless these symptoms are caused by spreading leptomeningitis, brain puncture should be repeated. If puncture does not reveal pus, one may reasonably assume that the symptoms are caused by edema or encephalitis, and conservative treatment (p. 377) should be instituted.

In favorable cases usually two to four punctures will achieve a cure. The last puncture often reveals clear or yellow fluid. This is probably not cerebrospinal fluid but serous exudate originating in the abscess walls. This finding indicates cure of the abscess. If purulent exudate continues to fill the abscess cavity, the outlook is not good (p. 366), and it is often necessary to switch over to the open method. Occasionally, a few weeks after the operation, rarely later, general and focal brain symptoms reappear, indicating the formation of a new abscess. If at this time the operative site in the temporal bone or frontal sinus is entirely healed, it is advisable to tap the new abscess through a sterile field.

With the open method the most important complication is brain herniation, which may be primary or secondary (p. 336). The herniation usually occupies the mastoid and tympanic cavity or the frontal sinus, but this need not interfere with the after-care provided the operation on the temporal bone or frontal sinus was completed before exploration of the brain. After-care is concerned only with the brain herniation, not with the surgical cavity in the bone. Infections in the tympanic cavity or frontal sinus rarely cause an abscess in the bulging portion of the herniation. If they do, fluctuation of the herniation can be observed, and the abscess is incised or it ruptures spontaneously toward the surface.

Herniation caused by brain edema, which occurs frequently in children, may gradually increase in size for a month or six weeks. There is no rapid extension or marked hemorrhage or necrosis, and the surface of the herniation consists of smooth, anemic cerebral cortex. Systemic symptoms are not present, but there may be general brain symptoms, particularly papilledema. The herniation is covered with vaseline gauze and conservative treatment (p. 377) is instituted. In these cases convalescence may extend over two months and more.

With herniation caused by nonpurulent localized encephalitis the findings are the same as those for brain edema. When the encephalitis is virulent and progressive, for example, phlegmonous encephalitis, the herniation grows rapidly, sometimes in a period of hours. Soon hemorrhages occur both on the surface and in the fungus, and every dressing causes a new hemorrhage. In a short time necrotic spots appear and extend rapidly. The surface of the herniation no longer consists of smooth cerebral cortex, but has a rough surface which is hemorrhagic, necrotic or purulent. Systemic symptoms may or may not be present, focal brain symptoms are striking and increase rapidly in number and intensity. Treatment is the same as for herniation caused by brain edema, except that chemotherapy must be added. Resection of the fungus is of little value because it recurs rapidly as long as the encephalitis is active and virulent.

With meningitis the fungus does not reach considerable size.

Moreover, the systemic and meningeal symptoms permit correct diagnosis. The abscess rarely refills, nor does a new abscess form if the open method was correctly performed. Pulmonary complications must be managed according to the standard methods.

If during convalescence the ventricle ruptures into the abscess cavity, everything must be done to insure free drainage of cerebrospinal fluid. The foot of the bed must be elevated, and dressings should not interfere with the leakage from the ventricles. Some surgeons suggest the forcing of fluids, with intake ranging between 3,000 and 9,000 cc. in 24 hours, depending on the age and size of the patient. Chemotherapy must be liberally applied. In a case of temporal lobe abscess, a great amount of crystal-clear fluid escaped from a brain herniation, although a gross fistula leading into the ventricle was not found. The fluid saturated the dressing, and even the pillow was wet. Fluid mixed with air bubbles also filled the subarachnoid spaces covering the herniation. The patient was cured.

Conservative treatment.—Conservative treatment aims at (1) increase of natural resistance, (2) disinfection of the brain and meninges and (3) reduction of intracranial pressure. To increase natural resistance, rest in bed, a high caloric diet and frequent catharsis or colonic irrigations are essential. Blood transfusions should not be given because they may cause brain edema associated with extreme rises of temperature. Great care must be exercised to prevent pneumonia or cystitis, particularly in elderly patients. These complications are common with brain abscesses, as are pulmonary emboli and abscesses if a brain abscess is associated with sinus thrombosis. The effect of sulfonamides and penicillin on brain abscesses has not been thoroughly studied, although other surgeons and I have used them with satisfactory results. From a theoretical point of view one may assume that these drugs should aid in the prevention of spread of encephalitis or meningitis. Whether or not they encourage the formation of a capsule cannot be stated. Of great importance is the treatment of increased brain pressure. For this purpose, spinal punctures and hypertonic solutions are available. Spinal punctures have particu-

lar value in the treatment of herniations; the injection of hypertonic solutions has only a temporary effect, at best. X-ray treatment of herniations does not yield definite results. Occasionally I have punctured the ventricle through a herniation caused by brain edema. The puncture was well tolerated and reduced the herniation.

TEMPORAL LOBE ABSCESS

PATHOLOGY

The temporal lobe is most commonly involved in otorhinogenic brain abscesses. The infection may originate in (1) the temporal bone, (2) lateral sinus of the dura, (3) cavernous sinus or (4) sphenoid sinus. Because the temporal bone is of utmost importance, this section deals particularly with otogenous temporal lobe abscesses.

In thrombophlebitis of the lateral sinus the infection may travel along the vein of Labbé (Fig. 7, p. 18) to the temporal lobe. The diagnosis is made only exceptionally during life because these abscesses are usually small and form a short time before death and the symptoms are usually obscured by those of sinus thrombosis. In thrombophlebitis of the cavernous sinus the infection is carried to the temporal lobe along the middle cerebral veins (Fig. 19, p. 56). The clinical courses of the two foregoing types of temporal lobe abscess are similar. Infection of the sphenoid sinus may cause a temporal lobe abscess if the sinus is extremely large and extends lateralward to the pole of the temporal lobe (Fig. 24, p. 74). These cases are rare and from a practical point of view are not important.

As far as the temporal bone is concerned, the principal sources of temporal lobe abscesses are the tympanic and mastoid cavities, the apex of the petrous bone and the temporal squama. Infections of the inner ear almost never give rise to a temporal lobe abscess, and the petrous apex and osteomyelitis of the temporal squama are unusual sources. The tympanic and/or mastoid cavities often give rise to these abscesses. The infection travels almost exclusively through the tegmen tympani by contiguity or by

continuity. The entire tegmen, from the superior petrous angle down to the roof of the eustachian tube, or only a part of it, may be involved. Infection by contiguity seems to be more common and causes necrosis or a fistula of the tegmen. Necrosis involving

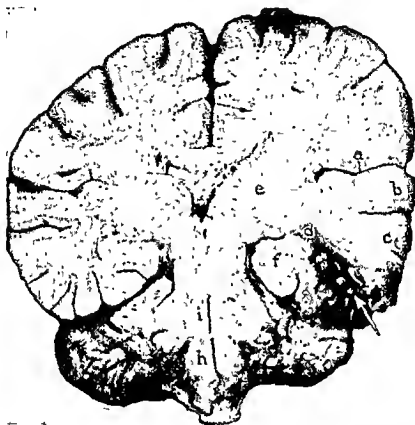


FIG. 81.—Acute abscess of temporal lobe. Note rupture into inferior horn of lateral ventricle (*d*) and enlargement of cerebral hemisphere on the involved side *a*, sylvian fissure; *b*, superior temporal gyrus; *c*, mesial temporal gyrus, *e*, internal capsule; *f*, cornu Ammonis; *g*, fusiform gyrus; *h*, medulla oblongata, *i*, pons. Arrow indicates opening of abscess toward the tympanomastoid cavity.

only a small area hidden in the roof of the eustachian tube may pass unnoticed, and the surgeon may think that the abscess was formed by infection by continuity. The dura of the middle cranial fossa may be normal or show external pachymeningitis. Among 21 cases of temporal lobe abscess I observed 10 in which the dura was definitely thickened and covered with granulations, and in

three of these there was a fistula. In seven cases the dura, as far as it was exposed at operation, was normal or presented slight changes, such as hyperemia or deposits of fibrin. Of these seven cases, two were caused by sinus thrombosis and in one case purulent encephalitis of the temporal lobe was discovered. In four there was an extradural abscess.

Otogenous temporal lobe abscesses primarily occupy the white matter of the third temporal gyrus. In a coronal section through the brain the abscess forms a cavity extending from the gyrus up and mesialward toward the inferior horn of the ventricle (Fig. 84), forward to the anterior boundary of the inferior horn, up toward the white matter of the second or, eventually, the first temporal gyrus and back toward the occipital lobe. Occasionally the abscess reaches considerable size. There is a recorded case of a child, aged 2½, who died of a temporal lobe abscess containing 250 cc. of pus. In such rare instances the abscess may extend up to the parietal lobe and postcentral gyrus, but never into the frontal lobe. If the temporal lobe abscess is associated with a brain-dura fistula the fistula usually pierces the third temporal gyrus.

Large pockets and diverticula are not uncommon with otogenous temporal lobe abscess. Multiple temporal lobe abscesses are believed to occur in about 0.5 per cent of the cases.

SYMPTOMATOLOGY

The systemic and general brain symptoms are discussed on page 352. This section deals with the ear and focal brain symptoms.

Ear symptoms—Temporal lobe abscesses may be caused by chronic or acute otitis and infections following mastoid operations. They are caused most often by chronic or acute otitis, with the former seeming to be of greater importance. Among 22 cases, I found chronic otitis in 14. Apparently the difference between acute and chronic otitis is not as great as many surgeons emphasize, for in acute otitis a temporal lobe abscess may appear after the infection in the tympanic cavity has undergone spontaneous

cure, obscuring the role of the acute otitis as a causative factor.

With chronic otitis cholesteatoma, particularly of the epitympanum, is of utmost importance. In a case presenting symptoms of brain involvement the finding of a cholesteatoma of the epitympanum definitely corroborates the diagnosis of brain abscess. Even more important is an acute exacerbation of a chronic ear infection (p. 190). An acute exacerbation may be noted in each phase of temporal lobe abscess. In the initial phase the cerebral symptoms are often obscured by the symptoms of the acute exacerbation, so that it may be impossible to determine the incipient stage of a temporal lobe abscess. In the quiescent phase acute exacerbations of slight degree may continue for a few days and do not appreciably affect the clinical course of the brain infection, although they may cause changes in the walls of the abscess. Finally, an acute exacerbation causes the quiescent stage to progress to the manifest stage, in which the cerebral symptoms usually obscure the symptoms of the acute exacerbation.

In the first two or three weeks, acute otitis does not cause formation of a brain abscess. Therefore it is the subacute (p. 113), not the acute, otitis which may be the source of the temporal lobe abscess. If in these cases there is a profuse discharge from the ear, if the drum membrane is red and slightly bulged and presents a nipple formation and if there is definite mastoiditis, every otologist will consider the ear a potential source of an intracranial complication. All of these symptoms may be absent, particularly in mucosis otitis (p. 103); nevertheless there may be an otogenous temporal lobe abscess. These infections of the tympanic cavity are often mistakenly called "latent otitis." These infections are not latent, because acute otitis cannot be considered cured until the local ear symptoms have subsided and until the patient has also regained his pre-infection physical status. When after the third week of acute otitis a patient complains of headache, occipital pain, tinnitus, dizziness or impairment of hearing, the otitis cannot be considered cured. It does not matter whether the drum membrane is red or gray, perforated or intact or whether there are definite mastoid symptoms or not. Unfortunately such patients

are often dismissed despite their symptoms. A few weeks later a brain abscess develops and an operation through a sterile field is usually performed. The operation is frequently successful because, of all otorhinogenous abscesses, those which develop after apparent cure of an infection of the tympanic cavity and mastoid process respond most favorably. Obviously brain abscesses of this type frequently could be prevented had a mastoid operation been performed at the proper time. This statement may seem to contradict the fact that temporal lobe abscesses may develop after a mastoid operation, but such cases commonly result from an inadequately performed simple mastoid operation. After a radical mastoid operation a temporal lobe abscess is extremely rare. I have seen only two cases.

In one case there was postoperative atresia of the external canal, and beyond the atresia was an accumulation of pus.

In the other case radical mastoid and sinus-jugular operations were performed. Two years later the patient complained of dizziness and occipital pain. There was a small amount of secretion in the tympanic cavity, but the labyrinth on the involved side was not excitable and the cerebrospinal fluid was cloudy and sterile. The operation did not reveal any pathology, but at autopsy the temporal lobe was found to be adherent to the dura which had been exposed two years before. There was an abscess in the temporal lobe. Since the temporal bones were not examined microscopically the pathogenesis of the abscess is not clear.

The more frequent incidence of temporal lobe abscesses after simple mastoid operations is apparently due to the fact that this operation is not standardized. Extension of the operation depends large on the pneumatization of the temporal bone, leading to errors in technique. Two technical errors are especially common. (1) Infected cells are left in the petrosal angle or zygoma, and the inflammation remains active in the cell walls and may cause an infection of the brain. (2) A simple mastoid operation is performed in the presence of subacute otitis. This accomplishes wide drainage of the mastoid cavity but not of the tympanic cavity, which is drained only through the narrow channel of the mastoid antrum. This drainage is sufficient when the

drum membrane is perforated and the tympanic cavity does not contain adhesions or granulations, in other words, when the operation is performed in the third or fourth week of acute otitis. In subacute otitis or acute recurrent otitis the tympanic cavity, particularly the epitympanum, may be partially filled with adhesions or granulations which interfere with drainage of pus through the antrum. Therefore, pus may be retained in the tympanic cavity, particularly when the perforation of the drum membrane is closed. Simple drainage of the mastoid antrum is, therefore, not sufficient. In all such instances the lateral wall of antrum and epitympanum should be removed (p. 122, footnote).

Focal brain symptoms.—These symptoms are local and remote. The former include disorders of speech and defects of visual fields, hearing and smell. Disorders of speech are found with 87.5 per cent of temporal lobe abscesses on the left side in right-handed persons. The size of the abscess has no appreciable effect on the manifestation of aphasia.

Although Head's doctrine of aphasia²¹ is not generally accepted, it serves the purpose of the otolaryngologist, who usually is more interested in establishing the diagnosis of intracranial complication than in analyzing the type of aphasia. According to Head, an organic disease in the speech area of the brain may cause four different types of speech defects.

1. Verbal aphasia. "The characteristic manifestations . . . consist mainly of inability to discover the exact form of words and phrases necessary to perfect external and internal speech, together with want of power to transform them into written characters." The patient understands speech which he hears or reads. The expression of numbers is poor, yet the relation of coins of different values is understood. This type of aphasia is usually caused by a lesion of the third frontal gyrus, in front of the anterior gyrus. The lesion is usually associated with hemiplegia or brachioptegia.

2. Syntactic aphasia. This is a more or less profound disorder "of the internal balance of a word as an orderly rhythmic expression." The patient talks rapidly but in jargon or paraphasia. He tends to omit prepositions, conjunctions and articles; polysyllabic words are shirred

²¹H. Head.: *Aphasia and Kindred Disorders of Speech* (Cambridge, England: 1926).

or poorly pronounced. He remembers or understands words but not phrases. The lesion is usually in the center of the first temporal gyrus, beneath the foot of the postcentral gyrus; associated symptoms are defects of the visual fields and perhaps uncinate attacks.

3. Nominal aphasia. In this type there is lack of power to designate objects by name. Although the patient possesses plenty of words he cannot apply them properly, particularly nouns. He paraphrases, confounds the words and uses mimetic expression. There is difficulty in understanding and executing oral or printed commands. He does not comprehend what he reads to himself, and spontaneous writing is poor, although he may write somewhat better on dictation. He does not properly identify letters, coins and numbers, and arithmetical problems are solved with difficulty. He can sing without words and knows whether music is correctly played by others, but he cannot read musical notes. This syndrome, which many neurologists consider a sensory aphasia in retrogression, is caused by a lesion in the angular gyrus, posterior third of the upper temporal gyrus and adjacent parietal area in front.

4. Semantic aphasia. "These defects are characterized by lack of recognition of the full significance of words and phrases apart from their immediate verbal meaning. The patient fails to comprehend the final aim or goal of an action initiated spontaneously or imposed on him from without." Some neurologists consider this not aphasia proper but an agnostic disorder. It is caused by a lesion in the area between the postcentral gyrus and the occipital lobe.

Verbal aphasia is more or less identical with the motor or expressive aphasia of the old nomenclature; the other types are varieties or phases of sensory aphasia.

Otogenous temporal lobe abscesses do not cause motor aphasia. Motor aphasia in a patient with an ear infection is due to a cerebral lesion independent of the ear infection, such as encephalomalacia, encephalitis and metastatic abscesses, or tumor of the frontal lobe or is caused by otogenous meningitis, not by a temporal lobe abscess.

A man, aged 55, with long-standing syphilis and deafness, had traumatic acute otitis on the left after removal of cerumen. In the fourth week of the otitis there were chills, fever about 102.2 F. and mastoid symptoms. At operation only hyperemic mucosa and a little serous exudate were found, but a day later motor aphasia was noted. Three days after operation there was a phlegmon of the left side of

the neck and chest, probably originating in the jugular bulb. The cerebrospinal fluid was clear. There were slight rigidity of the neck and a suggestion of Kernig's sign. The next day the patient died. Autopsy revealed purulent meningitis, with principal accumulation of pus in the anterior part of the sylvian fissure and area of the third frontal gyrus. There was no temporal lobe abscess.

Even though otogenous temporal lobe abscesses do not cause motor aphasia, they do affect the emissive component of speech. Occasionally patients who have recovered from temporal lobe abscesses have slow and scanty speech, sometimes with stuttering or stammering.

A child, aged 4 years, with a large temporal lobe abscess on the left side, displayed complete muteness which improved very slowly after operation. Even here, scrupulous analysis revealed that the muteness was due to sensory, not motor, aphasia.

Of the several types of sensory aphasia, nominal aphasia is most significant of temporal lobe abscess. It does not show uniform characteristics. One patient simply hesitates in designating objects by name. Another cannot name an object when he visualizes it but can if he makes contact with the object with other sense organs (optic aphasia). Another cannot understand nouns the perception of which requires associated action of the acoustic and optic centers (acoustic-optic aphasia). for example, he does not understand spoken words such as clouds, rainbow, streaming flags and similar expressions. Or he cannot name objects on contact by sense of smell or taste. Another cannot name countries, streets or towns (topographic aphasia). All these types of nominal aphasia are of utmost importance from a theoretical point of view, but for the otolaryngologist the finding of nominal aphasia per se is sufficient to warrant certain conclusions.

Aphasia with temporal lobe abscess sets in gradually; sudden onset is rare. Therefore, it may be missed in the incipient phase unless the surgeon is especially alert. Not infrequently aphasia increases after drainage of the abscess.

A man, aged 41, with chronic otitis on both sides and acute exacerbation on the left, presented the incipient phase of nominal aphasia.

He designated some objects correctly after some hesitation; other names, such as coffee cup, bunch of keys and dial plate, he could not express. For a third group of names he used paraphrases: Bell, "to ring", towel, "to wipe off." Reading was good and there was no paraphasia. The following day a temporal lobe abscess on the left side was drained. The day after operation he did not speak spontaneously, but examination revealed the following.

Understanding of spoken phrases.

How do you do?—Well, it isn't too bad (continued by paraphasia).

How did you sleep?—Well, well, told before.

Did you sleep well?—Yes.

Have you headache?—No.

What is your age?—(paraphasia).

How do you do?—Well, von see—God—not too bad.

What is your name?—(paraphasia using many numbers [a few minutes before his arithmetic was examined]).

What is your age?— 3×6 is 11.

Have you children?—Yes.

How many?—(correct)

Is your wife alive?—A fourth.

Are your parents alive?—Yes

What is the age of your parents?—Seven.

Where are your parents living?—Very well.

Recognition and naming of common objects.

Key—(paraphasia).

Bunch of keys—(no answer, tinkling of the keys did not elicit the word).

Nurse—(paraphasia)

Clock—(correct)

Pencil—(paraphasia)

Book—(paraphasia).

Reading: He read printed matter, exhibiting marked paraphasia. Written words he could not read, but he read what he had written himself. He did not understand what he was reading to himself.

Reading of numbers.

23—(correct).

73—(correct).

155—15 15

326—Three and twenty-six.

1746—One thousand seven forty-six.

Mental arithmetic.

$5 \times 5 = 25$.

3 × 7—21.

5 × 18—(no answer; he wrote the question as follows: 18 × 5=540).

Writing to dictation:

I am lying in bed—(wrote unintelligible words which sounded somewhat like the dictated phrase).

Today is a beautiful day—(correct)

That is a pretty bell—(correct).

The room is painted white—(correct, except the word "painted").

Twelve days later the patient died without regaining normal speech. The onset of the aphasia could not be determined.

This case demonstrates the rapid progress of the speech disorder after surgical drainage of the abscess. Frequently, however, surgical drainage improves the aphasia, although it may be weeks or even months before speech is restored since, in general, sensory aphasia does not recede as rapidly as motor aphasia.

An interesting condition is nominal aphasia with right-sided temporal lobe abscess in a right-handed patient. There is no satisfactory explanation. In some of these cases there is latent left-handedness; in others the aphasia may be due to contralateral hydrocephalus. The latter theory is entirely hypothetical.

A widespread opinion is that nominal aphasia in a right-handed patient with otitis media on the left side indicates the presence of temporal lobe abscess. In other words, with acute or chronic otitis on the left and nominal aphasia the temporal lobe should be explored. This is not wholly correct. Disregarding cases in which nominal aphasia is caused by a vascular disease of the temporal lobe or by a tumor, nominal aphasia may be due not only to a temporal lobe abscess but to subdural empyema, meningitis, extradural abscess or thrombosis of pial veins in the middle cranial fossa. Therefore, nominal aphasia with an ear infection does not permit definite conclusions concerning the appropriate type of surgery.

Defects of the visual fields are more common than the records suggest, because in drowsy patients the fields cannot be examined. Even if the mental state is clear it is not always feasible to hold the patient's attention for the time required for the examination. Nevertheless, in all suspected cases of temporal lobe abscess the

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Encephalitis

ENCEPHALITIS is an inflammation of the brain which may spread over wide areas without producing localized abscesses.

A distinction is usually made between purulent and nonpurulent encephalitis, although from the standpoint of etiology and pathology this distinction is not invariably practical. In fact, certain micro-organisms, for example, those causing influenza and typhoid fever, may cause either nonpurulent or purulent encephalitis. Observations in experimental infections of the brain also do not justify a sharp distinction between purulent and nonpurulent encephalitis. On the other hand, certain types of encephalitis, including epidemic encephalitis, polioencephalitis haemorrhagica superior and the bulbar type of poliomyelitis, invariably fail to cause the production of even a microscopic amount of purulent exudate.

Many neuropathologists do not distinguish between acute brain abscess and purulent encephalitis. They use the term "encephalitis" only for the nonpurulent type, and use the terms "purulent encephalitis" and "brain abscess" promiscuously. To the otolaryngologist, brain abscess means a gross accumulation of pus which requires surgery, whereas purulent encephalitis implies purulent or hemorrhagic-purulent exudate with only microscopic extension. Even though it advances to formation of one or several small abscesses, the encephalitis remains the primary lesion. In these cases surgery on the brain is useless. Microscopically, purulent encephalitis causes dilatation of the blood vessels, which

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visual fields should be examined, even though only an approximation with the finger is possible. In many cases there is homonymous hemianopia without involvement of the macula. In others there are only partial defects of the fields. Defects of the upper portion are especially common; defects of the lower portion are unusual. Partial defects usually recede when the abscess is cured; complete hemianopia frequently persists. Alexia (disturbance of reading despite good vision) has been noted with temporal lobe abscesses.

Defects of the visual fields are usually caused by a lesion of the optic radiation of Gratiolet. The optic radiation originates in the anterior quadrigeminal body, optic thalamus and, especially, the lateral geniculate body and forms a broad bundle of fibers. This bundle constitutes the lateral wall of the posterior and inferior horns of the lateral ventricle and runs into the optic centers at the mesial surface of the occipital lobe. When a temporal lobe abscess extends toward the lateral ventricle, as it usually does, the abscess or the surrounding encephalitis must necessarily involve either the entire optic radiation, causing hemianopia, or parts of it, causing partial defects of the visual fields. Recently it was emphasized that visual field defects are caused not only by a lesion of the optic radiation but also by a temporal pressure cone (p. 55) pressing on the tractus opticus on the same side or by obliteration of the posterior cerebral artery which carries blood to the optic centers.

Although nominal aphasia may occasionally be caused by an infection of the subdural spaces exclusively, any defects of the visual fields suggest that the cerebrum, not the meninges, is involved. In a patient with an ear infection, defects of the contralateral visual field are fairly strong evidence of cerebral involvement but do not necessarily establish the diagnosis of brain abscess.

In a young girl, a radical mastoid operation was performed on the right. Eight years later she suddenly became unconscious. Examination showed a small amount of secretion in the radical mastoid cavity, the cerebrospinal fluid was clear, but there was a defect in the left upper

quadrant of the visual field. Puncture of the right temporal lobe did not reveal pus. After tapping she felt well. When she was examined several months later, the field defects had not changed. Nevertheless, there was not the slightest indication of a brain abscess. An exact diagnosis could not be established, although several findings indicated the possibility of a brain tubercle.

Several reported cases are believed to prove that a temporal lobe abscess occasionally causes deafness on the opposite side, but there is no evidence that this is true. It is more likely that contralateral deafness is caused by a meningeal infection and subsequent infection of the contralateral inner ear. In a few cases of temporal lobe abscess anosmia was discovered on either the ipsilateral or the contralateral side. As with deafness, whether the anosmia is peripheral or central in origin cannot be stated.

The remote focal brain symptoms may be divided into (1) motor symptoms, (2) cerebellar symptoms and (3) symptoms of the cranial nerves. They are caused by various pathologic changes, including brain edema, contralateral hydrocephalus, displacement of the striate body, corpus callosum, septum pellucidum and lateral and third ventricles beyond the midline, and brain herniations.

Among motor symptoms, hemiparesis and occasionally hypesthesia are found on the contralateral side, and, less frequently, hemiparesis on the ipsilateral side. Contralateral hemiparesis is caused either by edema of the internal capsule or by a temporal pressure cone pressing on the cerebral peduncle on the ipsilateral side. Ipsilateral hemiparesis is a perplexing finding and may cause grave errors in diagnosis. It was my misfortune to make a diagnosis of *left-sided frontal lobe tumor* in a case of a large temporal lobe abscess on the right side because the patient's principal symptom was marked hemiparesis on the right. Ipsilateral hemiparesis with temporal lobe abscesses is probably caused by the same mechanism that causes ipsilateral hemiparesis with temporal lobe tumors. In both conditions protrusion of the temporal lobe down through the tentorial hiatus (p. 55) and shift of the brain stem toward the contralateral side must tend to thrust the contralateral cerebral peduncle against the sharp dural edge of

the tentorium, thus causing ipsilateral hemiparesis. Both ipsi- and contralateral hemipareses usually subside with cure of the abscess. The pathogenesis of contralateral monoparesis, particularly that involving the facial nerve, is not clear. Edema of the striate body is probably the cause of rigidity of the contralateral extremities and of mimetic palsy, occasionally found in these cases.

Twitching of the face, leg or arm and exaggeration of the tendon reflexes on the contralateral side are probably caused by brain edema. Brain edema may also cause deviation of the eyes and of the head to the involved side. Deviation of the eyes may or may not be associated with paralysis of lateral movement of eyes to the contralateral side. Jacksonian attacks and generalized epileptic convulsions are not common with temporal lobe abscesses; apparently they occur more frequently after recovery.

The statement that a temporal lobe abscess does not cause cerebellar symptoms, and vice versa, is too dogmatic. It is true that cerebellar abscesses rarely cause symptoms of temporal lobe involvement, although there are cases on record in which cerebellar abscesses caused nominal aphasia and paraphasia. In two cases of cerebellar abscess I noted defects of the visual fields. However, as previously mentioned, these findings are not common. More frequently, cerebellar symptoms are noted with temporal lobe abscesses, including spontaneous second degree nystagmus to the involved side, past-pointing with the contralateral arm, falling backward and to the contralateral side and vertical nystagmus, which occurs especially when the abscess ruptures into the ventricle.

Of the cranial nerves, the oculomotor nerve is often involved, causing various symptoms such as mydriasis on the involved side; unilateral ptosis, ptosis and mydriasis, ptosis, mydriasis and paresis of the internal rectus muscle, ptosis, mydriasis and paresis of the superior and internal rectus muscles, and complete oculomotor palsy. The commonest is mydriasis on the involved side, which occasionally can be increased by irrigation of the ear with cold water. Rupture of the abscess into the ventricle may cause bilateral mydriasis. Pupillary reaction is usually normal, rarely it

is sluggish. Only in the terminal stage of meningitis is pupillary reaction occasionally absent. Autopsy may reveal marked flattening of the oculomotor nerve. A temporal pressure cone causing downward displacement and compression of the oculomotor nerve against the edge of the petroclinoid ligament has already been mentioned. Pupillary symptoms may also be caused by compression of the quadrigeminal body. Paresis of the superior oblique muscle is apparently not common.

Patients with a temporal lobe abscess may complain of pain in the eyes and forehead. The pain is continuous but may fluctuate in intensity. There is no anesthesia or hypesthesia, and the corneal reflex is normal. Some neurologists attribute this type of pain to pressure of the abscess on the first branch of the trigeminal nerve. Whether or not this explanation is correct cannot be definitely stated. Abducens paralysis and paralysis of the hypoglossal nerve on the ipsilateral side are not common. Some otologists claim that temporal lobe abscess may cause necrosis of the inner ear from pressure on the internal auditory artery. I have never made such an observation either with temporal lobe abscesses or with acoustic tumors, which exert more pressure on the artery than do temporal lobe abscesses.

PROGNOSIS

According to the literature the mortality rate of temporal lobe abscesses is between 60 and 70 per cent. My experience includes 32 temporal lobe abscesses. In one group I acted as consultant only; in a second group I performed the operation or assisted at the operation but had no part in the after-care, and in a third group I managed both operation and after-care. Of the 32 abscesses, four were found at autopsy. Among the remaining 28 cases, 19 patients died (68 per cent) and nine were cured (32 per cent). This mortality rate is appallingly high. However, these cases were seen over a period of more than 25 years and were managed under various unfavorable circumstances. For one thing, the operation was performed whenever the diagnosis was made. Thus, in a considerable number of the cases the operation was

performed in the acute phase, which eventually would have become quiescent had no operation been performed. In another group the operation was done in the terminal phase. In several cases the brain abscess was associated with such severe infections as sinus thrombosis and labyrinthitis or severe systemic diseases such as arteriosclerosis and toxic leukopenia. Moreover, the surgical technic formerly used by the writer was not adequate. Finally, a considerable number of fatalities were caused not by brain abscess or meningitis but by pulmonary infection. In view of these facts, the statistics picture the prognosis for temporal lobe abscesses managed under exceedingly unfavorable conditions. They cannot be compared with statistics on chronic abscesses operated on in the quiescent phase, frequently after spontaneous cure of the infection of the tympanic cavity.

TREATMENT

Operation consists primarily of surgical removal of the entire tegmen. Regardless of whether or not the tegmen shows gross pathologic change, it should be removed from the superior petrosal angle to the eustachian tube. A simple mastoid operation does not provide sufficient space for drainage of a temporal lobe abscess, so that even with acute otitis the simple mastoid operation should be enlarged by removing part of the lateral attic wall, or a conservative radical operation should be performed if a low epitympanum does not permit the saving of the "bridge" of the mastoid antrum. When the tegmen is removed part of the temporal squama the size of a silver dollar is also removed (Fig. 85). The operation on the temporal bone should be completed before the brain is entered unless the patient is in a coma or there is difficulty in breathing. In the presence of these complications part of the tegmen is rapidly removed and an attempt immediately made to drain the abscess. Completion of the bone operation can be postponed.

For the brain operation a new set of instruments is prepared. The procedure of choice is the open method (p. 373). However, several conditions may interfere with the use of the typical tech-

nic. The changes in the dura must be considered. If the dura is grossly normal and there are no adhesions between dura and brain, a cross-incision is made and the four dural flaps are reflected (Fig. 86). When after incision of the dura a large amount of cerebrospinal fluid escapes from the subdural spaces, it is unlikely, although not impossible, that there is a brain abscess and further procedure should be postponed. The finding of external

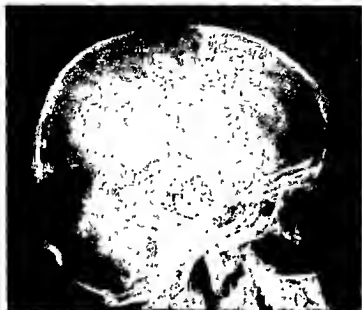


FIG 85.—Bone defect after operation for temporal lobe abscess.

pachymeningitis without dural adhesions does not interfere with this technic, whereas the finding of localized necrosis or of a dural fistula requires certain modifications. Both necrosis and fistula may be due to subdural empyema or brain abscess (pp. 134 and 140). In the presence of localized necrosis a cross-incision is made in the necrotic area exclusively, but the brain is punctured only if the diagnosis of brain abscess is definite. If the diagnosis is uncertain puncture of the brain should be postponed regardless of whether the dural incision causes primary brain herniation or not. If there is a dural fistula an attempt should be made to enlarge it provided there are no adhesions between dura and brain. If adhesions encircle the mouth of the fistula, the next step de-

depends on the disease causing the fistula. The technic for management of a fistula caused by internal pachymeningitis is discussed on page 140. If a brain abscess is the cause, the open method is not suitable, because a severing of the adhesions would encourage spread of the infection. Either simple puncture



FIG. 86—Operation for temporal lobe abscess through the infected field (open method) (By L. Bergmann.)

and suction of the abscess must be done through the brain-dura fistula or the open method must be used remote from the fistula. If the dura above the tegmen tympani is firmly adherent to the cerebral cortex the open method cannot be used and must be replaced either by the closed method or by the open method performed in the area of the temporal squama.

After the dura is incised the next step depends on whether or not a primary herniation (p. 335) occurs. If there is no primary herniation the cerebral cortex may present a normal appearance,

or it is anemic, the meningeal veins are dilated and there may or may not be a necrotic spot and eventually a cortical fistula. The latter changes indicate chronic brain abscess, and the open method with typical technic can be used. If the cerebral cortex is grossly normal, an exploratory puncture should be performed if clinical findings strongly indicate brain abscess. Otherwise con-



FIG. 87.—Temporal lobe abscess. 1, Primary herniation of brain, covered by delicate epidermis. 2, Two months later, showing disappearance of herniation.

servative measures should be tried and the result of the operation observed.

To puncture the temporal lobe, the cannula pierces the base of the temporal lobe in the area of the third temporal, or fusiform, gyrus and advances slowly up and mesialward toward the inferior horn of the ventricle for a distance of 3-4 cm. If within this distance the cannula enters the ventricle and clear cerebrospinal fluid escapes through the cannula, one may be almost certain that there is no abscess of the temporal lobe. Otherwise the ventricle would be compressed and the cannula could not enter it. Aspiration of normal brain tissue through the cannula is also a

strong indication of absence of a brain abscess, because brain tissue near an abscess shows edema or encephalitis. In these circumstances no more punctures should be performed. If, however, puncture does not permit definite conclusions and the clinical condition suggests brain abscess, two more punctures may be performed: one directed up and forward toward the pole of the temporal lobe, and one directed up and backward toward the occipital lobe. The first puncture is the more important. If one of these punctures reveals pus the open method can be applied.

Management of primary brain herniation (Fig. 87) caused by an acute abscess, and occasionally by a chronic abscess, is more difficult. Sometimes nature solves the problem, as when the abscess causes a fistula of the cerebral cortex or the brain protrudes with such force that the cortex bursts. In these instances the fistula should be enlarged to create proper drainage. The real problem is maintenance of drainage. The introduction of tubes into the abscess is not effective because soft tubes are compressed by edema and rigid tubes irritate the edematous brain and are rapidly expelled. Even gauze is apparently irritative, causing an increase of edema. For these reasons, repeated puncture of the herniated brain is the method of choice. In the presence of an acute abscess spinal punctures are not particularly effective, because they must not be repeated too frequently and because not more than 1-3 cc. of fluid can be withdrawn at each puncture.

If after incision of the dura primary brain herniation occurs but there is no brain fistula, the herniated brain should be punctured provided the clinical symptoms strongly indicate brain abscess. If the puncture reveals pus, management is the same as for spontaneous brain fistula. If no pus is revealed, conservative measures should be instituted and further changes of the herniation carefully observed.

FRONTAL LOBE ABSCESS

PATHOLOGY

Frontal lobe abscesses are caused by infections of the paranasal sinuses. Metastatic abscesses originating in a lung infec-

tion and traumatic abscesses do not come in the scope of this volume. Among the paranasal sinuses, the frontal sinus is the most frequent source of infection; the others are of minor importance except the ethmoids, the infection of which occasionally causes necrosis of the roof and then, by infection by contiguity, a brain abscess. Brain abscesses caused by infections of the maxillary or sphenoid sinuses are extremely rare. With maxillary sinus infection the orbit or pterygoid plexus may become involved and serve as a pathway for infection of the brain. With sphenoid infections a brain abscess may originate from necrosis of the planum sphenoidale, infection of the hypophysis or cavernous thrombosis.

The primary infection of the frontal sinuses may be either acute or chronic. The pathogenesis of frontal lobe abscesses is similar to that of temporal lobe abscesses originating in infections of the tympanic cavity. In the latter the tegmen tympani serves as pathway; in the former the posterior wall or, in the presence of a deep orbital recess, the superior wall of the sinus may allow the infection to travel to the brain by contiguity or by continuity. Infection by contiguity is more common, in both acute and chronic frontal sinusitis, occurring in about 57 per cent of cases. Infection by contiguity is more frequent with chronic infections, and infection by continuity with acute sinusitis. Infection through the bone by contiguity invariably causes distinct changes in the dura, including external pachymeningitis, extradural abscess or adhesions between brain, dura and bone and, eventually, necrosis of the dura or brain-dura fistula. Infection through the bone by continuity leaves the dura grossly normal and bulged, although in some cases there is external pachymeningitis or closed extradural abscess (p. 91), or even necrosis of the dura, although the bone fails to present gross changes.

Occasionally frontal sinus infection is associated with an abscess of the contralateral frontal lobe. The pathogenesis is not always clear. In some cases there is asymmetrical extension of the frontal sinuses, one sinus being in contact with both frontal lobes (Fig. 24). Infection of the larger sinus may give rise to an abscess

of the contralateral frontal lobe. In other cases frontal sinusitis causes external pachymeningitis which extends over the poles of both frontal lobes, and this, in turn, causes an abscess of the contralateral frontal lobe. Or an infection of the frontal sinus on one side may extend to the opposite sinus without causing conspicuous symptoms, and infection in the second sinus may cause a brain abscess. The formation of brain abscess in the contralateral frontal lobe must be considered even in cases of traumatic brain abscess.

A boy, aged 12, fell into a bush and pushed a twig into his right nostril. Immediately after the injury there were fever, headache, pain in the nose and hemorrhage from the nose. He was treated with antipyretics. When he was hospitalized three weeks later there was evidence of cerebral involvement. Examination revealed a piece of wood embedded in granulations in the right nostril. There were positive Babinski and Oppenheim signs on the left and facial paresis and hemiparesis on the left. Diagnosis was frontal lobe abscess on the right. Autopsy, however, revealed a frontal lobe abscess on the left side. Microscopic examination offered an explanation of this finding: the wood had pierced the uppermost part of the nasal septum, the lamina cribrosa on the left, and had caused a frontal lobe abscess and severe ethmoid infection on the left. It is noteworthy that there was persistent mydriasis on the left, the importance of this symptom was not properly evaluated.

Brain abscess occurs in, at most, 25 per cent of cases of rhinogenous osteomyelitis of the skull, acute osteomyelitis with protracted course (p. 96) being the principal source. Two different pathologic processes must be considered. In the first, the infection of the frontal sinus causes both brain abscess and spreading osteomyelitis, in the second, the sinusitis causes osteomyelitis which, in turn, causes a brain abscess. The former is more common. In these instances the brain abscess is a complication not of osteomyelitis but of the frontal sinus infection and is always found in the frontal lobe. The following case proves the practical importance of distinguishing the two types.

A boy, aged 16, became acutely ill with fever to 105 F., severe frontal headache, swelling of the right side of the forehead, edema of

the right eyelids, malaise, nausea and chills 14 days after swimming. About five weeks later there were convulsions, weakness, drowsiness and a large swelling over the right frontoparietal region. When I saw him for the first time, Oct. 2, 1943, there were edema of the right side of the forehead and a tumor the size of an egg in the right frontoparietal region. The lateral portions of both lids on the right were edematous, red and slightly tender. Both pupils were narrow, on the left more than on the right, and there were bilateral papilledema of 2 D. and facial paresis on the left. Cerebrospinal fluid was normal.

On October 5, both frontal and ethmoid sinuses were operated on. The left frontal and ethmoid sinuses were normal. There was a fistula in the anterior wall of the right frontal sinus, and osteomyelitis extended beyond the boundaries of the sinus. A gutter was chiseled into the bone from the lateral wall of the right orbit to the left part of the frontal squama above the left frontal sinus. The right side of the frontal squama, part of the right malar bone, the right supraorbital arch and the entire right frontal sinus were removed. The exposed dura was normal but definitely bulged. After the operation the patient continued to complain of headache. Facial paresis increased and there was paresis of the left arm. On October 16, the osteomyelitic bone in the right frontoparietal area was removed and a large area of external pachymeningitis exposed. Again convalescence was unsatisfactory. In the right frontoparietal region the brain, covered by inflamed dura, bulged through the skull defect and did not show pulsations. Papilledema increased to 4 D., and a diagnosis of brain abscess or subdural empyema in the inferior part of the right anterior central gyrus was made. For this reason, on November 26, the dura in the right frontoparietal region was incised and the brain punctured without success. An enormous brain herniation (Fig. 74) developed, but repeated punctures through the herniation did not reveal pus. The patient died January 11. Autopsy revealed a small abscess of the right frontal lobe about 4.5 cm. from the frontal pole.

There was apparently a grave misinterpretation of the underlying pathology. Although the diagnosis of brain abscess was made, it was assumed that it had originated in the osteomyelitis of the frontoparietal region, since there was severe pachymeningitis and the clinical symptoms indicated involvement of the motor area on the right side. Autopsy, however, revealed the abscess in the frontal lobe, probably originating in the frontal sinusitis, although there was no external pachymeningitis behind the posterior wall of the frontal sinus. It can reasonably be assumed that the abscess would have been drained had the puncture been performed in the area of the right frontal sinus.

As was previously mentioned, brain abscesses caused by osteomyelitis of the skull are less common than those caused by frontal sinusitis. If spreading osteomyelitis causes a brain abscess it usually first causes thrombophlebitis of the superior longitudinal sinus or osteomyelitis of the temporal bone. The infection of the



FIG. 55—Frontal lobe abscess, with rupture into anterior horn of lateral ventricle (*h*). *c*, caudate nucleus, *d*, lentiform nucleus, *e*, anterior part of internal capsule, *f*, posterior part of internal capsule, *g*, posterior horn of lateral ventricle; *h*, occipital lobe. Arrow indicates opening of abscess toward the frontal sinus.

superior longitudinal sinus or temporal bone, in turn, gives rise to an abscess in the parietal, occipital or temporal lobe or cerebellum. Diagnosis of thrombophlebitis of the superior longitudinal sinus is discussed on page 235. Osteomyelitis of the temporal bone is an insidious infection and does not necessarily cause pain or distinct changes in the drum membrane. Therefore, thorough examination of the temporal bones should be undertaken in all cases of spreading rhinogenous osteomyelitis of the skull associated with abscess in the temporal lobe or cerebellum. Rarely, spreading osteomyelitis of the skull causes brain abscess without

involving either the superior longitudinal sinus or the temporal bone.

The shape and direction of extension are not as uniform with frontal lobe abscess as with temporal lobe abscess because the posterior wall of the frontal sinus, the principal port of entry to the frontal lobe, varies more in size than does the tegmen tympani, the principal port of entry to the temporal lobe. In frontal lobe abscess the frontal sinus is usually medium size or large. For this reason, the abscess may originate at various sites in the frontal lobe and may even form in the contralateral frontal lobe. However, in general, frontal lobe abscesses occupy the white matter of the superior and, eventually, the middle frontal gyrus (Fig. 88). These areas are adjacent to the posterior wall of the frontal sinus. The abscess may extend back toward the anterior portion of the internal capsule, while encephalitis may involve the posterior portion of the internal capsule, which contains the supranuclear fibers of the facial nerve and muscles of the extremities. The abscess approaches the anterior horn of the lateral ventricle (Fig. 88) from a forward, downward or lateral direction. If there is a brain-dura fistula, it is behind the posterior wall of the frontal sinus in abscesses originating in the frontal sinus; it is above the ethmoid roof in abscesses originating in the ethmoid, and it is in the planum sphenoidale in abscesses originating in the sphenoid. Frontal lobe abscesses apparently cause diverticula more often than do temporal lobe abscesses. Occasionally a diverticulum extends through the corpus callosum into the frontal lobe on the other side. Multiple abscesses also are apparently more common in the frontal than in the temporal lobe. There may be one or even more abscesses in each frontal lobe.

SYMPTOMATOLOGY

Among otorhinogenous abscesses the diagnosis of frontal lobe abscesses is most dependent on systemic and general brain symptoms. Often there are no others. The nasal and focal brain symptoms are sometimes helpful but have less clinical value than systemic and general brain symptoms.

Nasal symptoms.—Frontal lobe abscesses occur with acute or chronic infections of the frontal sinus and after operations on the frontal sinus. Frequently an injury to the forehead is reported in the history. Statistics compiled from the literature and personal experience indicate that chronic infections caused 32 per cent of brain abscesses and acute and subacute infections, 68 per cent. Other rhinologists, however, have recorded contrary findings.

In infections of the paranasal sinuses the differential diagnosis of acute infection and acute exacerbation of chronic infection cannot be as exact as in infections of the tympanic cavity, because the sinuses cannot be directly inspected. Therefore, the differential diagnosis must be based on the history and on the finding of polyps and hypertrophy in the nasal cavity in chronic cases. The history is not always dependable, and polyps and hypertrophy may not be present in cases of chronic infections. Despite the uncertainty of diagnosis, so many of the cases in my statistics point to an acute infection as the source of frontal lobe abscess that an erroneous diagnosis cannot possibly be inferred in all. Thus it appears that acute infections of the frontal sinus cause frontal lobe abscesses more often than do chronic infections, which is in definite contrast with the findings in temporal lobe abscesses. The following reasoning may explain the difference.

Chronic sinusitis which is eventually a source of brain abscess differs in several ways from the chronic otitis apt to cause brain abscess. (1) The chronic otitis usually involves the tympanic cavity in early childhood and continues for several years; chronic infections of the frontal sinus begin in a later period of life, because in early childhood there are no frontal sinuses. (2) The chronic sinus infection is not often related to contagious diseases of childhood, but is due to acute infection following a common cold or to repeated acute infections which became chronic because of obstruction to drainage, such as a septal deviation, mucosal hypertrophy or a curled turbinate. Brain abscess from chronic otitis caused by an obstruction to drainage, such as

adenoids or hypertrophy of the lymphatic tissue in the tube, is rare. (3) The principal difference is cholesteatoma, which occurs frequently in the tympanic cavity and is extremely rare in the frontal sinus. Since cholesteatoma is the most common source of brain abscess, chronic infections of the frontal sinus necessarily cause brain abscesses less frequently than does chronic otitis. (4) An acute exacerbation of chronic otitis and one of chronic sinusitis must be evaluated differently. Chronic otitis causing bone absorption advances slowly toward the labyrinth or meninges as long as there is a discharge. Acute exacerbation indicates an acceleration of the advance whether or not pus is retained. Chronic sinus infections involving the underlying bone more often cause formation of new bone than absorption of bone. Therefore, an acute exacerbation does not necessarily accelerate advance of the infection toward the meninges, because ordinarily there is no slow progress toward the meninges before the exacerbation. However, an acute exacerbation of chronic sinusitis often causes total occlusion of the sinus ostium and thus retention of pus, which is increased by polyps in the sinus. This may lead to bone absorption and may eventually cause a brain abscess. In other words, in intracranial complications due to chronic sinusitis retention of pus is more often the causative factor than the acute exacerbation, whereas in similar complications due to chronic otitis the acute exacerbation is more often responsible than retention of pus. This is not opposed to the finding of a patent nasofrontal duct with frontal lobe abscess, because retention of pus is usually relieved by the time the brain abscess develops.

In acute cases the rhinologic examination reveals normal conditions, if the empyema is enclosed in the sinus, or pus in the anterior part of the middle meatus which rapidly reappears when it is wiped off. The acute infection destined to cause a brain abscess is always severe, as suggested by the duration of the infection, orbital symptoms and bone involvement. The duration of acute sinusitis is uncertain. However, acute sinusitis which is properly drained does not last more than four weeks. When

acute sinusitis advances toward the meninges and brain, the symptoms continue longer despite proper drainage. The discharge of pus is not necessarily continuous; on the contrary, it may stop in due course, but headache and tenderness continue and surgery may show the sinus filled with polypoid tissue. Since cerebral symptoms seldom appear before the fourth week of the sinus infection, it is actually not the acute but the subacute sinusitis which causes the brain abscess.

Orbital symptoms consist of lid edema, particularly of the upper lid, edema of the conjunctiva, eventually slight protrusion of the eyeball, displacement of the eyeball, usually down and outward, and occasionally fistula in the mesial angle of the upper eyelid. These symptoms are nearly always noted with a frontal lobe abscess caused by an acute sinus infection. However, they may already have subsided if the patient is seen first in a late stage of the brain abscess. Frequently the lid edema progresses to a lid abscess and a simple incision of the lid is performed without entering the sinus. This operation is useless, because invariably a fistula forms which does not close in the presence of a brain abscess.

In chronic sinus infections the rhinologic examination does not reveal characteristic changes. The findings are the same as in simple chronic sinusitis, i.e., pus and, eventually, polyps or hypertrophy of the turbinates. Orbital symptoms occur in about 50 per cent of the cases.

Frontal lobe abscesses are rarely caused by operations on the paranasal sinuses unless osteomyelitis develops postoperatively. I have the impression that acute osteomyelitis associated with a frontal lobe abscess does not show the unlimited spreading characteristic of acute osteomyelitis which is not associated with brain abscess.

Facial brain symptoms—True symptoms of the frontal lobe are rarely noted with rhinogenous frontal lobe abscesses. There are mental and psychic changes, such as irritability, drowsiness and abrupt changes from cheerfulness to melancholia, but these are caused by the intracranial hypertension. Compulsive grasping

and groping are rare with frontal lobe abscesses. Spontaneous nystagmus and disturbances of body balance and of smell are noted occasionally. More common symptoms are (1) convulsions, (2) pareses and (3) pupillary changes.

Convulsions are more frequent with frontal than with temporal lobe abscesses. At the onset there is only a twitching of the facial muscles on the contralateral side. As the disease advances the arm and, finally, the leg become involved, causing a typical jacksonian attack. In advanced cases general epileptic convulsions may occur. With a frontal lobe abscess and acute osteomyelitis, the patient frequently reports that there were convulsions at the onset. This is particularly likely if osteomyelitis sets in with fulminating symptoms, such as high fever, chills and drowsiness. I have never observed convulsions at the onset of the infection and so cannot give an analysis of them. But certainly such "convulsions" do not necessarily indicate brain abscess. Usually they occur only once or twice, then disappear, regardless of whether or not the infection is destined to cause a brain abscess. Convulsions caused by the brain abscess, not by osteomyelitis, become manifest in a later phase of the infection.

With the appearance of jacksonian attacks, paralyses are noted. The muscles of the contralateral side of the face are the first to be involved. Since the facial paralysis is supranuclear in origin, only the mouth is involved, the branches for the palpebral fissure and forehead acting fairly normally. Facial paralysis is seldom marked. Often only the nasolabial fold becomes indistinct, and the angle of the mouth is lower, as noted particularly *when the patient is quiet or asleep*. Tendon reflexes on the contralateral side are exaggerated and pathologic reflexes (p. 274) are present. Facial paralysis is soon followed by paralysis of the contralateral arm; finally, the leg may become paralyzed, completing the syndrome of hemiplegia. With frontal lobe abscesses hemiplegia usually develops in distinct phases, except for facial paresis which often passes unnoticed in the initial phase. Paralysis of arm and leg usually appears suddenly. If the patient recovers, use of the extremities is often, but not always, restored.

Pupillary changes are not frequently reported; apparently they often pass unnoticed. Nevertheless, mydriasis on both sides or on the involved side, less frequently on the contralateral side, and sluggish reaction or fixed pupils are comparatively common.

PROGNOSIS

The mortality rate of frontal lobe abscesses is between 60 and 70 per cent. Of nine cases in my experience, eight were caused by head injury and one by an infection of the frontal sinus with acute osteomyelitis. Five patients recovered. As was the case for temporal lobe abscesses, these statistics picture the prognosis under most unfavorable circumstances. It is the associated osteomyelitis which renders the prognosis serious and not, as in temporal lobe abscesses, the associated sinus thrombosis or pachymeningitis.

TREATMENT

The open method is the procedure of choice in frontal lobe abscesses. The operation on the frontal sinus should be performed according to the technique of Biedel, i.e., the inferior and anterior walls should be removed. If the sinus is small, parts of the frontal squama must also be removed to gain access to the abscess. This operation leaves a marked deformity of the face, except when the sinus is small, but no other method permits complete inspection of the posterior sinus wall. The dura is then exposed by removal of the posterior wall. If there is a deep orbital recess, the roof of the recess, which is a continuation of the posterior sinus wall, must also be removed. After the operation on the frontal sinus is completed, the open method can be applied with the same technique as for temporal lobe abscesses (Fig. 89).

Brain puncture is performed at a distance of 1-1.5 cm., and not higher than 2 cm., from the base of the frontal lobe. The cannula is directed straight back, or back and lateralward, to penetrate the brain for a distance of 1.5 cm. If the puncture is made at a height of more than 2 cm., the cannula should not be pushed farther than 3-4 cm.

A special problem is offered in the treatment of a frontal lobe abscess associated with acute osteomyelitis. Since osteomyelitis frequently develops before the brain abscess, there is time to cure the osteomyelitis before manifestation of the brain abscess. However, inefficient attempts to arrest the osteomyelitis

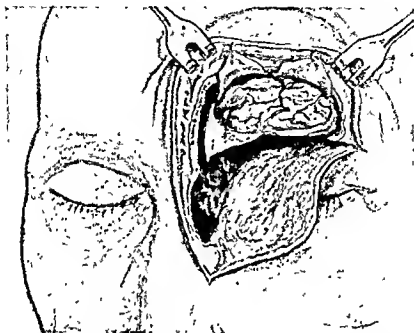


FIG. 89.—Operation for frontal lobe abscess through the infected field (open method). (By L. Bergmann.)

may leave the surgeon with a brain abscess associated with active osteomyelitis. In such instances the procedure depends entirely on the clinical findings. If there are signs of impending meningitis or symptoms of marked intracranial hypertension, the brain abscess must be drained and the operation for osteomyelitis postponed. If there are neither signs of meningitis nor symptoms of intracranial hypertension, management of the osteomyelitis should be undertaken before drainage of the brain abscess is attempted, because the diagnosis of a brain abscess frequently may be definitely established after the surgical procedure on the osteomyelitic skull.

CEREBELLAR ABSCESS

PATHOLOGY

Cerebellar abscesses originate in infections (1) of the petiolabyrinthine cells, (2) of the labyrinth and (3) of the lateral sinuses.

An infection to the mastoid antrum sometimes travels through the mesial wall to the part of the petrous bone located between the dura of the posterior cranial fossa and the semicircular canals. In temporal bones with reduced pneumatization the petrous bone may contain marrow spaces; in well pneumatized temporal bones it contains the pericanalicular cells (p. 111) which drain into the antrum, petrosal angle and Trautmann's triangle. An infection extending from the antrum into this part of the petrous bone involves the pneumatic cells in cases of acute otitis and the marrow spaces in cases of chronic otitis. An infection established in the posterior part of the petrous bone may extend into (1) the semicircular canals, (2) the subarcuate fossa and (3) the posterior cranial fossa. Infection extending close to the semicircular canals leads to serous labyrinthitis. If it invades the semicircular canals there is usually localized labyrinthitis, i.e., labyrinthitis involving only the area of the semicircular canals, not the cochlea. Rarely, diffuse purulent labyrinthitis results. Extension into the subarcuate fossa is also rare. It occurs principally with chronic and occasionally with subacute otitis. Along the connective tissue and blood vessels of the subarcuate fossa the infection travels toward the dura of the posterior cranial fossa and, eventually, into the cerebellum. Although these two pathways are not commonly involved, extension of the infection toward the dura of the posterior fossa is comparatively frequent. In such instances the infection absorbs the bone and by contiguity causes deep external pachy meningitis or a deep extradural abscess of the posterior fossa (p. 96). The abscess is invariably of the closed type (p. 95) and often a forerunner of cerebellar abscess. Obviously the infection in the posterior part of the petrous bone may extend

simultaneously in different directions, for example, into the semi-circular canals, causing localized labyrinthitis, and into the posterior fossa, causing cerebellar abscess.

The second source of cerebellar abscess is purulent labyrinthitis. About 7 per cent of cases of labyrinthitis are complicated by cerebellar abscess. Diffuse purulent and latent labyrinthitis are the principal offenders. In these types the infection travels either along the vestibular aqueduct, causing sacculus empyema (p. 130) and then cerebellar abscess, or along the internal auditory meatus. In other cases the purulent exudate in the inner ear absorbs the bony capsule, then the petrous bone, and invades the posterior fossa. The infection often travels along two or three pathways simultaneously. Although the labyrinth serves comparatively often as a source of cerebellar abscess, infection of the petrous bone without involvement of the inner ear is no less important.

Thrombophlebitis of the lateral sinus as source of cerebellar abscess is discussed on page 156. The infection seldom travels directly through the cerebral wall of the sinus into the adjacent cerebellum. If it does, a cortical abscess of the cerebellum results. A subcortical cerebellar abscess is usually due to internal pachymeningitis of the posterior cranial fossa caused by thrombophlebitis of the lateral sinus. In one of my cases thrombophlebitis of the superior petrosal sinus caused a cerebellar abscess; the petrosal sinus infection was caused by a cholesteatoma of the tympanic cavity.

Cerebellar abscesses present various shapes. The abscess often forms a cleftlike cavity between the cerebellar cortex and the white matter (Fig. 90) and is almost invariably located beneath the superior surface of the cerebellum immediately below the tentorium cerebelli. The cleftlike cavity may extend from the anterior edge of the cerebellum to the posterior edge and from one cerebellar hemisphere to the other. The abscess does not extend deep into the white matter and never involves the dentate nucleus. The infection may extend toward the cerebellar cortex, creating diverticula of the abscess which

may simulate multiple abscesses. However, multiple abscesses are not common in the cerebellum. The cleftlike type of cerebellar abscess, which rarely forms a capsule, for convenience may be called a "flat" cerebellar abscess (Fig. 90).

A second type of cerebellar abscess involves the white matter lateral to the dentate nucleus. The abscess forms an oval or spherical cavity (Fig. 91) which may reach considerable size within the white matter of one cerebellar hemisphere exclusively. The dentate nucleus is usually involved, and a capsule is often noted. Like flat abscesses, these so-called spherical abscesses also tend to form diverticula suggesting multiple abscesses.

Brain and brain-china fistulas (p. 341) are less common with cerebellar than with cerebral abscesses because of the briefer duration of cerebellar abscesses. If a fistula does form it is localized either on the superior surface or on the anterior edge of the cerebellum. If the fistula opens at the superior surface pus may accumulate between the cerebellum and the tentorium.

In one case of this type I observed an extension of the infection along the blood vessels of the tentorium. This caused a second accumulation of pus above the tentorium. The infection extended between the two occipital lobes of the cerebral hemispheres, involved the calcarine fissure and caused defects of the visual fields.

If the fistula opens on the anterior edge of the cerebellum the infection usually extends into the lateral pontile cistern (p. 38) and causes meningitis. Ruptures of the abscess into the fourth ventricle are not common.

SYMPTOMATOLOGY

Ear symptoms.—Like temporal lobe abscesses, cerebellar abscesses are caused by acute and chronic otitis and by infections following mastoid surgery. Chronic otitis is generally considered the principal source. However, among 12 personal cases, six were due to acute otitis and six to chronic otitis. In one case of the second group there was a cortical abscess. These statistics indicate infection usually extends into the lateral pontile cistern (p. 31)



FIG. 90.—Flat type of cerebellar abscess. *a*, anterior edge of cerebellum; *b*, posterior edge of cerebellum; *f*, brain fistula, *d*, dentate nucleus. (After Grabscheid)

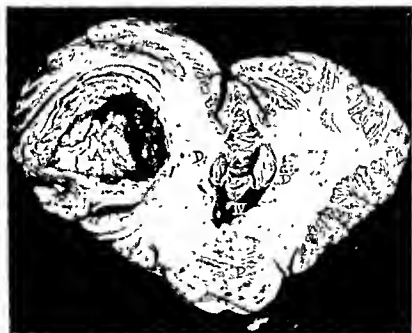


FIG. 91.—Spherical type of chronic cerebellar abscess (*A*). *D*, dentate nucleus; *IV*, fourth ventricle; *P*, pons. Note inspissated pus on the abscess walls and enlargement of cerebellar hemisphere on the involved side.

cate that the preponderance of chronic otitis is not as great as some surgeons claim.

The symptoms of cerebellar abscess usually appear in the subacute phase of otitis media. Pneumococcus type III infections are of great importance. The otoscopic findings are the same as in temporal lobe abscess, and, as in the latter, the perforation of the drum membrane is often closed by the time the abscess is diagnosed.

In one of my cases the drum membrane was yellow and there was no perforation, although at operation periphlebitis of the lateral sinus, bulbous thrombosis, external pachymeningitis of the middle cranial fossa and a cerebellar abscess were discovered. Friedlaender's bacilli were found in the cerebrospinal fluid and the pus of the mastoid process. In the cerebellar abscess, which presented a capsule, gram-negative bacilli with a mucous capsule, probably Friedlaender's bacilli, were found.

Exceptionally, a cerebellar abscess forms early in the course of acute otitis.

A man, aged 48, had influenza. Four days later there was acute otitis on the right due to *Streptococcus hemolyticus*, associated with listeria in the external canal and facial paralysis. Three weeks later a simple mastoid operation was performed. Twenty-four days after onset of the acute otitis, the symptoms of a cerebellar abscess appeared, and at operation a brain-dura fistula was discovered in the posterior cranial fossa. Thirty-nine days after onset of the acute otitis the patient died. At autopsy a spherical abscess was discovered in the right cerebellar hemisphere the duration of which was a little more than 15 days. Microscopic examination of the temporal bones was not made.

In chronic otitis, cholesteatoma of the tympanic cavity is the principal source of cerebellar abscesses. The acute exacerbation has the same importance as in meningitis and temporal lobe abscess. Infections following mastoid surgery have equal importance in the pathogenesis of both cerebellar and temporal lobe abscesses.

In one of my cases two operations on the mastoid were performed by another otologist. The type of operation was not exactly discovered. The result of these operations was incomplete atresia of the external

auditory canal and progressive osteitis of the walls of the mastoid antrum. Two months after the last mastoid operation the patient died of a cerebellar abscess caused by deep external pachymeningitis of the posterior cranial fossa.

The ear changes with cerebellar abscesses are frequently associated with involvement of the labyrinth, facial nerve and lateral sinus, singly or together. In the past, infections of the labyrinth were considered the principal source of cerebellar abscess. This is no longer true, for, owing to the reduced incidence of purulent labyrinthitis, infections of the petrous bone apparently cause cerebellar abscess as frequently as do infections of the labyrinth. Not every type of labyrinthitis threatens the cerebellum. Diffuse purulent labyrinthitis caused by cholesteatoma of the tympanic cavity is the principal offender. Diffuse labyrinthitis caused by acute otitis threatens the leptomeninges more than the cerebellum. Two types of diffuse labyrinthitis may be caused by chronic otitis media: a manifest type, associated with intense vertigo and nausea, spontaneous nystagmus, disturbance of equilibrium, deafness and nonexcitability of the labyrinth, and a latent type, associated with slight dizziness, first degree nystagmus to either side or to the contralateral side only, deafness and nonexcitability of labyrinth. The latter causes cerebellar abscesses more frequently than does the former.

Facial paralysis is common with cerebellar abscess. Among 12 personal cases, facial paralysis was noted in eight—five cases of acute otitis and three of cholesteatoma. The pathology of facial paralysis is apparently not unique in these cases and depends on the time of its appearance. If the paralysis develops before or with the symptoms of brain abscess, it may be caused by the infection of the tympanic cavity. If it appears in the late phase of cerebellar abscess, it is caused by an infection of the pontile cistern. In these instances there may also be trigeminal involvement, resulting in facial neuralgia or corneal hypesthesia on the ipsilateral side. The cisternal infection itself may be caused by a brain fistula through which pus escapes from the cerebellar abscess to the cistern or by external pachymeningitis which ex-

tends to the cistern. Therefore, facial paralysis in the late phase of cerebellar abscess is an unfavorable symptom indicating the initial stage of terminal meningitis. The paralysis is always peripheral in origin; i.e., all three branches on the side of the ear infection are involved.

In cerebellar abscess caused by sinus thrombosis the symptoms of sinus infection obscure those of the abscess. Nevertheless, even in these instances diagnosis of the abscess is often possible because of symptoms which cannot be attributed to the sinus thrombosis, including drowsiness, constant vomiting, rapid prostration and cerebellar symptoms. The association of sinus thrombosis and cerebellar abscess does not always imply that one is the result of the other. Frequently progressive osteitis of the petrous bone causes, in succession, sinus thrombosis, localized or diffuse labyrinthitis, extradural abscess and cerebellar abscess.

Focal brain symptoms.—As in temporal lobe abscess, a distinction should be made between local and remote symptoms, although this is somewhat arbitrary as far as several remote symptoms are concerned.

The principal local symptom is ataxia, which indicates a disturbance of muscle co-ordination. The patient is asked to close his eyes and place his index finger on the tip of his nose, to touch with the index finger of one hand the index finger of the other hand or to touch his knee with the other heel. With cerebellar abscess none of these movements are properly performed on the involved side. Babinski designated a particular type of cerebellar ataxia *asynergie cerebelleuse*, meaning disturbance of the synergistic action of different groups of muscles in the performance of complex movements. For example, if the patient raises the leg to step forward he does not move the body correspondingly forward. Or, if the patient bends backward he does not flex his knees normally, but keeps the legs extended. Or, if the patient is recumbent and wishes to sit up he raises his legs but not his body. Other cerebellar symptoms are: *adiadokokinesis*, or the inability to perform contradictory movements in a rapid succession, such as pronation and supination of the hand, *bradyphasia*,

or slow and slightly slurred speech, and tremor of the foot and hypotonia on the involved side.

Of particular importance are disturbances of equilibrium and errors in the pointing test, because they may occur with infections of both the cerebellum and the labyrinth. The equilibrium is examined in Romberg's positions (Fig. 92). The patient stands erect, with feet together and arms hanging naturally. Romberg's position should not be confused with Romberg's sign, which is

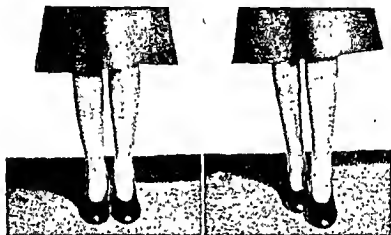


FIG. 92.—Romberg's position (left) and heel-toe position (right).

particularly suggestive of tabes dorsalis and in which there is unsteadiness when standing with eyes closed but none with the eyes open. With cerebellar abscess there may be unsteadiness when the eyes are open as well as when they are closed. The patient falls either backward or toward the involved side, without reference to the direction of nystagmus and position of the head, whereas the patient with an infection of the labyrinth falls in the direction of the slow component of nystagmus and the direction of falling changes with change of position of the head. If with cerebellar abscess spontaneous disturbances of equilibrium are not marked, they can be increased by reducing the base. This is done by placing the heel of one foot in front of the toes of the other foot or by placing the heel of one foot next to the great toe of the other foot (Fig. 92). With marked disturbance falling is

apparent even when the patient stands with the feet well apart.

The pointing test yields useful information when properly performed. There are two techniques. In neither should the patient exercise any optic control over his action; his eyes should be closed before he undertakes the test. In the first procedure he simply stretches his arms. With cerebellar abscess the arm on the involved side frequently deviates outward, whereas with labyrinthine disease both arms usually deviate in one direction. In the second procedure the patient sits in a chair, with his hands on his knees and the head in normal position. He is told to stretch the index fingers of both hands and to raise one arm about 1 M. When the arm is raised the examiner touches the patient's finger, regardless of whether or not the patient has deviated. This prevents the patient's making a correction during the test. If the patient does not make contact with the examiner's finger he realizes that he did not raise his arm correctly and attempts to correct an eventual deviation. A normal person will follow a perpendicular line on raising his arm, whereas a patient with cerebellar abscess frequently deviates. In some instances only the arm on the involved side deviates outward, and in others both arms deviate to the involved or contralateral side. Apparently the presence of the permanent deviation has clinical significance; the direction of deviation and involvement of one or both arms do not allow definite conclusions. The two techniques do not always yield identical results, so both must be applied.

Cerebellar gait, which is common with cerebellar tumors and rare with cerebellar abscess, is characterized by a profound disturbance of the gait mechanism. Occasionally a staggering gait is noticed: this is not necessarily caused by the cerebellar lesion *per se*, but may be due to vertigo and emaciation. Convulsions sometimes occur. They consist principally of tonic contraction of the muscles of the extremities and opisthotonos. The following incident is typical.

At 5.00 A.M. a patient with a cerebellar abscess on the left side suddenly screamed and turned rapidly to the right side, where he remained motionless for several seconds. At 7.00 o'clock he again

screamed and twisted his body so that his hips lay flat on the bed while the upper half of the body was twisted to the right with face down. His arms were flexed and rigid. The legs were extended and the feet flexed. There was no muscular twitching, just rigidity. The fit continued for about 30 seconds and was followed by stupor. At 8:00 o'clock the stupor continued but he answered questions and there was a coarse tremor of the left foot. The patient was immediately operated on and a cerebellar abscess on the left side was successfully drained.

These cerebellar symptoms may be striking or absent. Without venturing a definite statement, I believe that the symptoms are more conspicuous with the spherical than with the flat type of cerebellar abscess. Some surgeons claim that they are more marked with a cerebellar abscess caused by acute otitis (except those associated with sinus thrombosis) than with those caused by chronic otitis.

Among remote symptoms, nystagmus is of utmost importance, although in some cases nystagmus is not observed over a long period of observation. In the examination for spontaneous nystagmus the patient bends his head 30 degrees back, the upper eyelid is gently raised to permit observation of conjunctival blood vessels, and the patient looks at the examiner's finger held about 1 M. from the patient's eyes. The eyes should not be allowed to remain in the same position for more than one minute, because a slight nystagmus is readily suppressed by prolonged and voluntary fixation. Nystagmus is not a genuine cerebellar symptom; it is caused by pressure of the cerebellar abscess on the vestibular nuclei in the medulla oblongata. For this reason, it is occasionally absent with cerebellar abscess; furthermore, it may change in the same patient at different times. The term "cerebellar nystagmus" should be replaced by the term "nystagmus in cerebellar abscess," since nystagmus is not a cerebellar but a labyrinthine symptom.

The nystagmus with cerebellar abscess is rhythmic, consisting of a rapid and a slow movement of the eyes. The direction of nystagmus is recorded according to the rapid movement, although it is the slow movement which is controlled by the labyrinth. This apparently irrational nomenclature is practical, because the rapid

movement differs distinctly from voluntary ocular movements, which are never jerking, while the slow movement resembles the voluntary ocular movements. For this reason, the rapid movement is more conspicuous and more easily noted than the slow movement. Labyrinthine nystagmus may present different degrees of intensity. For example, nystagmus of first degree to the left is noticeable only when the patient moves his eyes to the left; nystagmus of second degree to the left is apparent when the patient looks to the left and straight ahead, nystagmus of third degree to the left is seen when he looks to the left, straight ahead or to the right. To evaluate spontaneous nystagmus it is necessary to distinguish cerebellar abscesses which are and are not associated with infection of the labyrinth. Without labyrinthine involvement the nystagmus is more often coarse than fine and more often horizontal than rotatory, and it is usually slow. It can be noted when the patient looks to the involved side only or when he looks to either side. Vertical nystagmus upward is not common. Sudden changes of direction of the nystagmus which occur over a few hours without apparent cause are significant. Occasionally a change is caused by a shift of position or by a particular position of head, or by the insertion of a tampon tightly into the retroauricular wound in the presence of exposed dura. There is always labyrinthine vertigo, but it may be slight while there is a distinct nystagmus.

If cerebellar abscess is caused by diffuse purulent labyrinthitis, say, on the right side, the labyrinthitis causes a horizontal and rotatory nystagmus of first to third degree to the left. Nystagmus caused exclusively by labyrinthitis gradually disappears in about two weeks, leaving behind a nonexcitable labyrinth. If marked nystagmus continues longer or shifts to the side of the nonexcitable labyrinth, a tentative diagnosis of cerebellar abscess can be made. This is Neumann's sign. It does not justify a definite diagnosis of cerebellar abscess because it may occur with an extradural abscess in the posterior cranial fossa, serous meningitis and intercurrent erysipelas, and occasionally even a temporal lobe abscess.

Not infrequently a forced position of the head and ocular deviation are noted. The ocular deviation is usually to the contralateral side and may or may not be associated with paresis of conjugate movement of the eyes to the involved side. In one of my cases the eyes deviated downward. Forced position of the head is not alike in all cases. Usually the head is turned toward the contralateral shoulder, or the chin is dropped toward the contralateral clavicle. The head is rarely turned toward the involved side. There may or may not be associated rigidity of the neck. Forced position of the head also occurs with deep extradural abscesses of the posterior cranial fossa (p. 101) and basilar meningitis, and I observed it in one case of purulent encephalitis of the temporal lobe. Abducens paresis, mydriasis and diminution of the corneal reflex on the involved side occur occasionally. Trigeminal neuralgia and trismus are rare. Tendon reflexes are frequently normal, although they are sometimes definitely diminished on one or both sides or are exaggerated and associated with a positive Babinski sign and diminution of abdominal reflexes.

PROGNOSIS

The prognosis for cerebellar abscesses is less favorable than that for cerebral abscesses. Some clinics admit to a mortality rate of about 100 per cent. Among 13 personal cases the mortality rate was 85 per cent. There are four principal causes of this high mortality rate. (1) Chronic abscesses are less frequent in the cerebellum than in the cerebrum. (2) Cerebellar abscesses are commonly associated with other serious infections, such as sinus thrombosis, progressive osteitis of the petrous bone, labyrinthitis and deep extradural abscess of the posterior cranial fossa. (3) Cerebellar abscesses are more likely to cause sudden respiratory paralysis than are cerebral abscesses. (4) Although cerebellar abscesses rarely rupture into the ventricles, meningitis from external infection or the associated infections of the sinus, dura, labyrinth and/or the petrous bone is common. Obviously the prognosis is more favorable when these complicating factors are

absent. Therefore, recovery from cerebellar abscesses which take a chronic course or follow the cure of acute otitis and mastoiditis is not uncommon, particularly in children.

TREATMENT

It seems to be the consensus that in cerebellar abscesses the open method performed through an infected field is not feasible, because there is insufficient space unless the lateral sinus is severed and or the semicircular canals are removed. This, however, may be done only when the lateral sinus and or the semicircular canals are infected before the operation. The open method is equally unsuccessful when performed through a sterile field. The flat type of abscess especially cannot be properly drained by the open method. Therefore the procedure of choice is the closed method.

If the lateral sinus is normal, the labyrinth active and the abscess is caused by osteitis of the petrous bone near the mastoid antrum, the dura in Trautmann's triangle, i. e., mesial to the lateral sinus, must be exposed. Regardless of whether the dura is normal or not, a nick incision is made and a cannula introduced (Fig 93). The cannula should be slightly curved like the trocar used to puncture the maxillary sinus. The cannula is pushed back toward the posterior edge of the cerebellum and simultaneously up toward the tentorium and somewhat mesialward toward the dentate nucleus. If no pus is discovered and puncture is repeated, the direction of puncture should not be changed except to direct the cannula somewhat lateralward instead of mesialward. Punctures lateral to the sinus should be performed only when Trautmann's triangle is very small because of marked bulging of the lateral sinus and jugular bulb or when the dura in the triangle is covered by a granulation layer so thick that it is not possible to identify the lateral sinus. Otherwise such puncture is not advisable because it strikes the abscess at a greater depth than puncture mesial to the sinus, it often passes below the abscess and it does not follow the route by which the infection entered the cerebellum. A difficult problem arises when an infection of the

petrous bone has caused a cerebellar abscess and deep extradural abscess of the posterior cranial fossa but has not involved the labyrinth. The labyrinth often must be sacrificed (p. 109).

When a cerebellar abscess is associated with sinus thrombosis, the sinus and then the jugular vein must be operated on first, unless the abscess requires emergency management. Since the

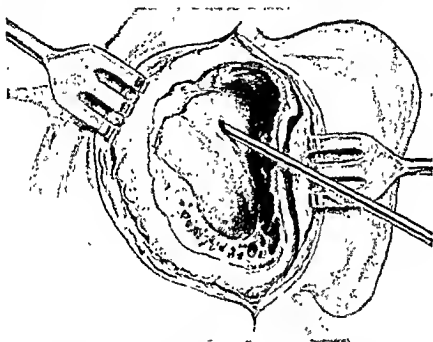


FIG. 93.—Operation for cerebellar abscess through the infected field (closed method). (By L. Bergmann.)

infection rarely enters the cerebellum through the mesial sinus wall (p. 409) and since a brain-dura fistula rarely opens into the sinus, the site of cerebellar puncture is the same as in cases without lateral sinus involvement. Ligation and severing of the sinus are not practical, because they may cause a rapid increase of brain pressure if the sinus is not involved and spread of the infection if the sinus is involved.

When the cerebellar abscess is associated with diffuse purulent labyrinthitis, the labyrinth operation (p. 109) must be performed before drainage of the abscess.

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Encephalitis

ENCEPHALITIS is an inflammation of the brain which may spread over wide areas without producing localized abscesses.

A distinction is usually made between purulent and nonpurulent encephalitis, although from the standpoint of etiology and pathology this distinction is not invariably practical. In fact, certain micro-organisms, for example, those causing influenza and typhoid fever, may cause either nonpurulent or purulent encephalitis. Observations in experimental infections of the brain also do not justify a sharp distinction between purulent and nonpurulent encephalitis. On the other hand, certain types of encephalitis, including epidemic encephalitis, polioencephalitis haemorrhagica superior and the bulbar type of polionmyelitis, invariably fail to cause the production of even a microscopic amount of purulent exudate.

Many neuropathologists do not distinguish between acute brain abscess and purulent encephalitis. They use the term "encephalitis" only for the nonpurulent type, and use the terms "purulent encephalitis" and "brain abscess" promiscuously. To the otolaryngologist, brain abscess means a gross accumulation of pus which requires surgery, whereas purulent encephalitis implies purulent or hemorrhagic-purulent exudate with only microscopic extension. Even though it advances to formation of one or several small abscesses, the encephalitis remains the primary lesion. In these cases surgery on the brain is useless. Microscopically, purulent encephalitis causes dilatation of the blood vessels, which

may or may not be thrombosed and which show conspicuous perivascular infiltration. The alteration in blood circulation causes hemorrhages and necrosis. The necrotic areas are often invaded by leukocytes and bacteria, forming small abscesses. These changes, suggestive of hemorrhagic-purulent encephalitis, extend gradually into an area of acute nonpurulent encephalitis which exists alongside the purulent encephalitis. In the latter area are newly formed capillaries, many of them surrounded by plasma cells, but there is no formation of purulent foci or hemorrhages. These observations indicate that the difference between purulent and nonpurulent encephalitis is one of intensity only.

Granting that all types of brain infections originate from localized nonpurulent encephalitis (p. 327), the original focus may give rise to the following types of inflammation of the brain: (1) nonpurulent encephalitis which may undergo spontaneous cure without becoming purulent, (2) nonpurulent encephalitis which becomes purulent and may advance further to (a) encephalitis phlegmonosa (p. 425), (b) acute brain abscess (p. 348), or (c) chronic brain abscess (p. 315).

From the standpoint of pathology, the three types of purulent inflammation differ only in rate and extension of spread, intensity of breakdown of the tissue and reaction of surrounding tissue. The rate and extent of spread are maximal in phlegmonous encephalitis, but of minor importance in chronic brain abscess. On the other hand, breakdown of tissue is a minor finding in phlegmonous encephalitis, but is the most conspicuous feature in chronic brain abscess. In phlegmonous encephalitis the surrounding tissue simply becomes necrotic without showing a definite reaction to the infection, whereas in chronic brain abscess the surrounding tissue forms a capsule to arrest the advancing infection. Acute brain abscess presents some features of both phlegmonous encephalitis and chronic brain abscess. It does not spread as rapidly and as extensively as phlegmonous encephalitis and the breakdown of tissue is more striking, but the reaction of the surrounding tissue is not as vigorous as that observed with chronic abscess.

OTOGENOUS ENCEPHALITIS

PATHOLOGY

The term "otogenous encephalitis" is applied to infections of the brain, both purulent and nonpurulent, believed to originate in the temporal bone. There is sufficient evidence to justify the assumption that purulent encephalitis may originate in infections

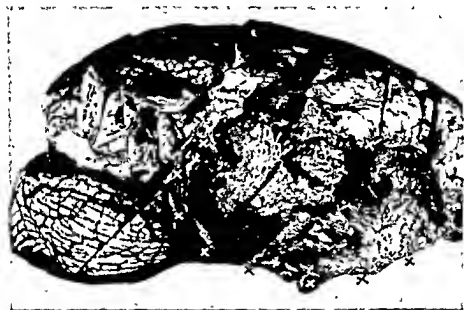


FIG. 94.—Large herniation of the brain in encephalitis phlegmonosa.

of the temporal bone or in otogenous infections of the dura or dural sinuses. In these instances the encephalitis advances like a phlegmon of the brain, and so is called "phlegmonous encephalitis" (Figs. 94 and 95). This type of encephalitis is not as common as brain abscess. I have seen only three cases. The pathogenesis is the same as that of brain abscess. Phlegmonous encephalitis is known to occur only in the cerebrum, although there is no reason to assume that it cannot occur in the cerebellum. Like brain abscesses, phlegmonous encephalitis may rupture into the ventricle and may cause terminal meningitis.

Whether or not nonpurulent encephalitis may be caused by

infections of the temporal bone is controversial. Theoretically, this eventuality cannot be denied, since, in principle, there is no difference between purulent and nonpurulent encephalitis. But



FIG. 95.—Coronal section through right occipital hole in a case of encephalitis phlegmonosa. *a*, small abscess cavity; *b*, hemorrhagic-purulent encephalitis; *c*, brain herniation. There is no abscess cavity that can be attacked by surgery.

from a practical point of view, nonpurulent encephalitis of otitic origin is somewhat problematic if it is considered a morbid entity. Autopsy observations are not available, because this type of encephalitis is not fatal. In the few cases of death from what seemed to be nonpurulent encephalitis, microscopic examination revealed changes which must be classified as phlegmonous encephalitis rather than nonpurulent encephalitis.

SYMPTOMATOLOGY

Phlegmonous encephalitis is caused by acute or chronic otitis or by otogenous sinus thrombosis. The temperature curve is irregular, but high fever is common, particularly before death. The pulse rate is also irregular. In the early phase temperature and pulse are related, but later the curves diverge. Leukocytosis is moderate or not present; there is a shift to the left and polymorphonuclears show toxic granulation. There is definite apathy, and the headache is intense unless decompression of skull has been performed. The cerebrospinal fluid is hemorrhagic, cloudy or clear and the cell and protein contents are invariably increased. Micro-organisms are found in the fluid only immediately before death.

Focal brain symptoms, such as hemianopia, aphasia and spasticity are present but fluctuate in intensity. Papilledema is absent or not marked. If decompression is performed, there is primary herniation of the brain (Fig. 94) which steadily increases. The illness lasts only several days and terminates in fatal meningitis.

There is no great difficulty in differentiating between phlegmonous encephalitis and chronic brain abscess because the striking systemic symptoms with the former are not seen with chronic abscess. The differentiation of phlegmonous encephalitis and acute brain abscess is more difficult. The following observations may be helpful. The course of acute brain abscess is not as rapidly downhill as that of phlegmonous encephalitis. Fever is not as high or as irregular in acute abscess as in phlegmonous encephalitis. The encephalogram in phlegmonous encephalitis is normal, but may show pathologic variations in acute brain abscess. Puncture of the brain gives positive results in acute abscess and negative results in phlegmonous encephalitis.

An accurate discussion of the symptomatology of otogenous nonpurulent encephalitis is not possible because autopsy studies are not available. The similarity of symptoms of serous meningitis, phlegmonous encephalitis and brain abscess and of non-

purulent encephalitis renders differential diagnosis extremely difficult. The following symptoms are believed to suggest non-purulent encephalitis.

1. The patient is not acutely ill, whereas patients with phlegmonous encephalitis or acute brain abscess are acutely ill.

2. There may or may not be high fever. In children it is nearly always found. Quiescent brain abscess does not cause high fever.

3. In adults the mental state may be slightly disturbed; in children there are invariably drowsiness, stupor and coma.

4. There is intense headache, and occasionally nausea and vomiting, but eye-grounds are normal or there is slight and temporary papilledema. This is contrary to observations in the hypertensive type of serous meningitis, in which papilledema is the principal symptom. The oculomotor nerve is often involved, usually on the ipsilateral side, occasionally on the contralateral side, causing mydriasis and eventually ptosis and loss of pupillary reaction.

5. There are cerebellar and temporal lobe symptoms. The former include tremor, adiadosokinesis and, eventually, ataxia. Of the temporal lobe symptoms, hemianopia is not usual, but aphasia is relatively common. It must be recalled that aphasia associated with ear infections is almost invariably the nominal type (p. 384). Motor aphasia indicates involvement of the frontal lobe. If this involvement is caused by encephalitis, the encephalitis is not likely to be of otitic origin even in the presence of an ear infection. The following case is an instructive example.

A man, aged 29, with chronic otitis on the left, became ill with a severe upper respiratory and bronchial infection on April 11. Four days later he suddenly presented motor aphasia, he stammered and his speech was not understandable, but he could understand. There were incontinence and such marked dizziness that he could not leave his bed. On April 23, Lasegue's sign was positive and there was slight rigidity of the neck. He fell and walked to the left. There were 7,000 leukocytes in the blood, and the cerebrospinal fluid showed 73 cells and a positive Pandy reaction. On the left side, chronic otitis was found without signs of acute exacerbation. The neurologic symptoms gradually subsided, but on June 10 he was delirious. The fundi were

normal. On June 17, there were central facial paresis on the right and an ataxia on the left. The patient was again delirious and presented a mixed motor-sensory aphasia with particular involvement of the emissive component of speech. For this reason, a temporal lobe abscess was considered and, on June 21, a radical mastoid operation was performed. The attic and antrum were filled with granulations, but the tegmen was normal and was left intact. After the operation the neurologic symptoms subsided but did not disappear entirely. The patient was improved when he left the hospital. There were no signs of brain abscess.

The motor aphasia was caused by involvement of the left frontal lobe, and it can reasonably be assumed that there was encephalitis. The absence of an acute exacerbation and the surgical findings indicated that the encephalitis was not otitic in origin but was probably caused by the influenza which preceded the development of neurologic symptoms. The improvement after operation was a coincidence, inasmuch as at an earlier date a similar improvement was noted without surgery.

6. There are monopareses, particularly of the facial nerve, or hemiparesis and, in children, jacksonian attacks. These occur on the side opposite the ear involvement. The appearance of motor symptoms on the ipsilateral side eliminates otogenous nonpurulent encephalitis from consideration. The pareses continue for a comparatively long time and well past the acute phase of the infection. This is contrary to the findings in the inflammatory type of serous meningitis (p. 289), in which the pareses and systemic symptoms of the acute infection usually subside simultaneously.

7. Meningeal symptoms are slight and the cerebrospinal fluid is normal or hemorrhagic. In the inflammatory type of serous meningitis the meningeal symptoms are more striking.

8. Encephalography yields normal results in nonpurulent encephalitis, whereas it discloses displacement of the ventricles in chronic and, at times, acute brain abscesses.

9. Puncture of the brain gives negative results in phlegmonous encephalitis and serous meningitis, but gives positive results in brain abscess.

10. Strange as it seems, little stress has been laid on the ear changes. According to the general pathology of intracranial com-

plications, one might suppose that otogenous nonpurulent encephalitis was caused especially by acute otitis due to an upper respiratory infection or an acute exacerbation of chronic otitis. However, in reported cases the symptoms of nonpurulent encephalitis developed most frequently in cases of chronic otitis without the slightest sign of acute exacerbation, or after well performed radical mastoid operations. These ear findings do not support the concept of the otogenous origin of encephalitis.

11. Nonpurulent encephalitis is not fatal, unlike phlegmonous encephalitis and brain abscess, which are fatal unless proper drainage is established.

Only a few reported cases of otogenous nonpurulent encephalitis presented all of the foregoing features. Apparently otogenous nonpurulent encephalitis, as a morbid entity, is rare; it is probably not as rare in association with meningeal infections. This observation does not refer to cortical encephalitis associated with any type of meningitis; but it does refer to internal and external pachymeningitis with which cerebral symptoms such as aphasia and, eventually, hemianopia (pp. 101 and 137) are seen. These symptoms certainly are not caused by the pachymeningitis *per se*. As the following case shows, at least in some cases they are caused by nonpurulent encephalitis associated with pachymeningitis.

A man, aged 30, in 1907 underwent a radical mastoid operation and jugular ligation on the left side. In 1914, an abscess formed in the upper part of the retroauricular incision. The abscess was incised and a fistula developed in this area. There were no other complaints.

In November, 1920, the right ear was normal. On the left side the cavity was partly epithelialized, partly covered with granulations. There was a fistula in the retroauricular incision the size of a pea. On November 11, plastic closure of the fistula was performed. The patient made an uneventful recovery and was dismissed. On December 9, he complained of dizziness and vomiting. On December 11, there were headache, dizziness, vomiting, nystagmus of second degree to the right and slight rise of temperature. On December 13, the temperature was normal. There was no dizziness or vomiting, but there were severe headache on the left side and an abundant discharge from the left

tympenic cavity and retroauricular fistula. The labyrinths were excitable and the neurologic examination gave negative results. On December 16, morning temperature was 102.5 F. He complained of intense pain in the forehead; the cervical spine was tender and there was slight rigidity of neck. There were some ataxia on the left side, nystagmus of second degree to the right and typical nominal aphasia. No papilledema was found. The middle cranial fossa was explored and marked edema of the temporal lobe was found. The meninges and superficial layer of the brain were hyperemic and there was no pulsation of the brain. Puncture did not reveal pus. The cerebrospinal fluid was cloudy and spinal pressure was increased. The following day the patient died.

Autopsy revealed a cavity of considerable size in the left temporal lobe filled with blood and debris. At several sites the cavity anastomosed with the inferior horn of the ventricle. There was no brain abscess. In the ventricle coagulated blood was found. The leptomeninges at the base were dull and there was subdural empyema over the left temporal lobe. Microscopic examination of the left temporal lobe revealed leptomeningitis with very slight involvement of the superficial layer of the cerebral cortex; in the deeper layers were several minute foci of encephalitis. Near the inferior horn of the ventricle was a small area of soft consistency. In this area the blood vessels were markedly dilated and some were ruptured. Marked perivascular infiltration, consisting of poly- and mononuclear cells, invaded the surrounding white matter. The myelin fibers in this area were destroyed. In the neighborhood of this area were several other small foci of encephalitis.

In this case there were subdural empyema and encephalitis of the left temporal lobe. The encephalitis must be classified as nonpurulent because there was no appreciable breakdown of tissue. There was no brain abscess. It can reasonably be assumed that the nominal aphasia was caused by the nonpurulent encephalitis, not by the subdural empyema. The etiology of the cerebellar symptoms was not clarified; it is probable that there was also nonpurulent encephalitis of the cerebellum.

It seems certain that nonpurulent encephalitis of the white matter of the temporal lobe may be associated with internal pachymeningitis of otitic origin and that the encephalitis may cause symptoms similar to those of a temporal lobe abscess. What remains to be proved is that otogenous nonpurulent encephalitis

of the temporal lobe is an autonomous morbid entity, i.e., without associated infection of the meninges.

PROGNOSIS

The prognosis for phlegmonous encephalitis is not favorable. All of my cases were fatal. The prognosis for nonpurulent encephalitis is good unless it is associated with a meningeal infection, although hemiparesis may last well beyond the acute phase of encephalitis. In some cases permanent changes of the mental state have been noted after otogenous nonpurulent encephalitis.

TREATMENT

No definite information is available concerning treatment of phlegmonous encephalitis of otitic origin. Simple decompression and x-ray treatment do not cure. Whether chemotherapy is effective is not known. In nonpurulent encephalitis the purulent focus in the temporal bone must be eliminated. Removal of the tegumen tympani and exposure of the dura is not necessary unless the tegmen is necrotic and there is external pachymeningitis. Chemotherapy may hasten definite cure.

PHARYNGEAL ENCEPHALITIS

It is fairly well established that there is a causal relationship between tonsillectomy and poliomyelitis, especially between recent tonsillectomy and bulbar poliomyelitis. Whether bulbar poliomyelitis is caused by an actual postoperative virus infection of the tonsillar bed or by a flare-up of a latent infection in the tonsils is not determined.

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